

FOOD SAFETY AND QUALITY CONTROL MEASURES

By

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DECLARATION

I, Aneena E. R. (2004-24-01) hereby declare that the seminar entitled "Food safety and quality control measures" have been prepared by me, after going through the various references cited here and has not been copied from my fellow students.

Vellanikkara

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CERTIFICATE

This is to certify that the seminar report titled **“FOOD SAFETY AND QUALITY CONTROL MEASURES”** has been solely prepared by Ancena, E.R (2004 - 24 - 01), under my guidance, and has not been copied from any seniors, juniors or fellow student's seminar reports.

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CONTENTS

Sl. No.	Title	Page No.
1.	Introduction	1
2.	Physical hazards	2
3.	Biological hazards	3
4.	Chemical hazards	9
5.	Food quality and control measures	19
6.	International food standards	22
7.	Conclusion	28
8.	References	29
9.	Discussion	31
10.	Abstract	

INTRODUCTION

Access to good quality food has been man's endeavor from the earliest days of human existence. Safety of food is the basic requirement of food quality. Food safety is a global challenge to meet the requirements of consumers with respect to hygienic and nutritional qualities. Food systems in developing countries are not always well organized and developed as in the industrial world. Therefore people in developing countries are exposed to a wide range of potential food quality and safety risks. Producing good quality and safe food is a pre requisite to successful and sustainable development of national agricultural resources, domestic and international food trade (Prakash, 2003). The changing consumer knowledge, behavioral as well as reliability of scientific merit on associated risk factors has brought the food safety issue in to a sharper focus

Terminologies related to food safety

Safety A substance is termed as safe, when it is practically certain that under conditions of use, no toxic injury will result

Food safety Is the assurance that damage or injury from the use of a food substance is impossible

Hazard It is the relative probability that a toxic substance can cause a toxic injury under conditions of use

Toxicity It is the capacity of a substance to produce harm or injury of any kind under any condition

Risk: Is expressed in the human population and is a function of a probability and severity of the adverse health effect that may occur due to hazard in food

Control measure Any action or activity that can be used to prevent or eliminate a food safety hazard or reduce it to an acceptable level

Quality assurance: It may be defined as a plant and systemic pattern of allocations necessary to provide adequate confidence that the product will perform satisfactorily in actual operation.

Major food safety issues of concern

- ❖ Physical hazards
- ❖ Biological hazards
- ❖ Chemical hazards

Physical hazards

Foods may contain hazardous substance such as stones, seeds, glass fragments, small bits of metal etc. These materials can become part of food from the natural environment in which they are grown or they may be contaminated during processing and packaging.

Main material of concern as physical hazards and common sources

Material	Injury potential	Sources
Glass	Cuts, bleeding, may require surgery to find and removal	Bottles, jars, light fixtures, utensils, gauge covers.
Wood	Cuts, injections, choking require surgery to remove	Fields, pallets, boxes, buildings
Stones	Choking, broken teeth	Fields
Metal	Cuts, Injections, require surgery to remove	Machinery, fields, wire
Insects & other filth	Illness, trauma, choking	Fields, plant post process entry
Bone	Choking trauma	Fields improper, plant processing
Plastic	Choking cuts, injection require surgery to remove	Fields, plant packaging
Personal effects	Choking, cuts, broken teeth, may require surgery to remove	Materials, pallets, employees

Measures to prevent physical hazards

Make sure your plant specifications for building design and operation are accurate and updated regularly

- Make sure your letters of guarantee for ingredients and product suppliers are accurate and update regularly
- Perform random visual examinations of incoming product and materials
- Use magnets and metal detectors to help find metal fragments that would be a physical hazard
- Use stone traps and bone separators to remove these potential physical hazards
- Keep all filtering and sieving/sifting device in good conditions of repair and maintenance
- Strictly follow glass protocol in the processing area
- Train employees to enable them to identify physical hazards.

Biological hazards

Biological hazards include bacterial, fungal, viral and parasitic organisms and their toxins. There are many microorganisms, which are pathogenic in humans, but relatively few are associated with food, these are known as food born pathogens. Diseases caused by these organisms are sometimes incorrectly called food poisonings. There are two types of food diseases from microbial pathogens. They are termed as infections and intoxications. Infections result from ingestion of live pathogenic organism, which multiply within the body and produce disease. Intoxication occurs when toxins produced by the pathogens are consumed. The majority of the health problems that are arising in the world over are due to consumption of foods contaminated with pathogens or microbially spoiled foods (Reghuramiah, 2001). Chakravarty (2003) observed the presence of *E.coli*, yeast, mould, salmonella, *sheigella*, *cholera vibrio*, *klebisella pneumoniae*, *pseudomonous acruigenosa*, *proteus mirabilis*, *proteus vulgaris*, *bacillus species etc* in Indian street food on a microbial analysis. The microbial quality of ready to consume snacks and beverages were examined for suitability for general consumption. (Rao, 1995) and the results are given below.

Microbial quality of some beverage foods used widely by the Indian consumers

Products	Pathogens found
Sugar cane juice (fresh)	<i>E. coli</i> , <i>Vibrio cholerae</i> , <i>Aspergillus flavus</i> , <i>Bacillus cereus</i>
Grape juice (fresh)	<i>Aspergillus flavus</i>
Flavoured milk	<i>E. coli</i> , <i>Yersinia</i>
Malted drink powders	<i>E. coli</i>

(Rao, 1995)

Identification of hazards in street foods of Vadodara, India revealed that high total *Mesophilic Aerobic Bacteria*, *S.aureus*, *E.coli*, *Salmonellas*, and *sheigella* (Sheth *et al*, 2005)

Salmonella

Salmonella causes one fifth of the human food poisoning. *Salmonella typhimurium* and *S enteritidis* are two commonly found species. *S typhimurium* is antibiotic resistant. The Center for Disease Control (CDC) reports that about 1.4 million people in US are affected by salmonellosis. These bacteria can be found in food products such as raw poultry, eggs, beef, and high protein foods. Symptoms usually begin 12-36hrs after ingestion and continue from 1-7 days. Diarrhoea, fever, abdominal pain, headache, etc are the major symptoms of salmonellosis.

Most people recover successfully from salmonellosis, but a few develop a chronic condition called Reiter's syndrome. This can last for months and years and can lead to arthritis. The symptoms are painful joints, irritated eyes and painful urination.

Salmonella typhosa is the causative organism of typhoid fever, which is characterized by fever, slow pulse rate, enlargement of spleen, rose spots on the trunk, ulcers in the intestinal tract and constipation. These bacteria are transmitted through contaminated water and food.

Botulism

Botulism is a rare but serious illness caused by toxin produced by *clostridium botulinum* bacteria, that grow in low acid foods like unprocessed canned foods its an anaerobic bacteria it can live inside sealed containers.

Symptoms -Double vision, drooping eyelids, slurred speech, dry mouth and difficulty in swallowing, and weak muscles. This will begin with in 18-36 hrs after eating. It may occur from 1-7 days. People who develop severe botulism , experience breathing failure and paralysis.

Streptococcus

Streptococcus are found in dairy products, fermenting juices, and organic matter containing sugars, this is the causative organism of scarlet fever, tonsillitis, purpual fever and sore throat, septicaemia, various skin and wound infections. To prevent this pasteurization of milk should be done

Staphylococcus

This most common food poisoning is due to *staphylococcus aureus* the symptoms occur with in 2-4 hrs after consumption. The organism grow well in between 12^oC - 44^oC. This poisoning is characterized by vomiting, diarrhoea and dehydration. This is mainly seen in cream pastries, salted meats and sausages

Shigellosis

Also called bacillary dysentery caused by *shigella* bacteria. Out breaks of shigellosis frequently occur in tropical or temperate climates. Sanitary handling of food, purification of drinking water, pasteurizations of milk, sanitary disposal of sewage, extermination of flies are the control measures.

Shigella can be infected from

- Eating food or drinking beverages contaminated by food handlers infected with *Shigella* who didn't wash their hands properly after using the bathroom
- Eating vegetables grown in fields containing sewage
- Eating food contaminated by flies which were bred in infected faeces

- Drinking or swimming in contaminated water

S. sonnei is the most common type of *Shigella* in developed countries, including the United States.

Symptoms

- Fever
- Tiredness
- Watery or bloody diarrhea
- Nausea and vomiting
- Abdominal pain

Symptoms usually begin within 2 days after being exposed to *Shigella*. Symptoms usually are gone within 5 to 7 days.

***E. Coli* infection**

Certain types of *Escherichia coli* bacteria, commonly called *E. coli*, can cause food borne illness. Harmless strains of *E. coli* can be found widely in nature, including the intestinal tracts of humans and warm-blooded animals. Disease-causing strains, however, are a frequent cause of both intestinal and urinary-genital tract infections. Several different strains of harmful *E. coli* can cause diarrhoeal disease. A particularly dangerous type is called enterohemorrhagic *E. coli*, or EHEC. EHEC often causes bloody diarrhoea and can lead to kidney failure in children or people with weakened immune systems.

Transmission

E. coli bacteria and its toxins have been found in

- Undercooked or raw hamburgers
- Alfalfa sprouts
- Lettuce
- Unpasteurized milk, apple juice, and apple cider
- Contaminated well water

Symptoms

E. coli O157:H7 toxin can damage the lining of your intestine and cause other symptoms including

- Nausea

- Severe abdominal cramps
- Watery or very bloody diarrhea
- Tiredness

You might develop low-grade fever or vomiting. Symptoms usually begin from 2 to 5 days after eating contaminated food and may last for 8 days.

Bacillus Cereus

Bacillus cereus is a facultative anaerobic microorganism. It is associated with two types of illnesses. Illness can either be associated with diarrhoea or vomiting.

A wide variety of foods including meats milk vegetables and fish have been associated with those pathogenic bacteria, which cause diarrhoeal type illness. The vomiting type illness is usually associated with starchy foods such as rice, potatoes and etc

Clostridium Perfringens

Most common forms of food poisoning is due to this micro organism. Usually contaminated from meats, eggs, gravies, and other protein foods. The illness is especially prevalent because of improper practice of keeping meats gravies warm. Good handling practice is more important in preventing this

Fungi

Fungi species produce mycotoxins. Aflatoxin, patulin are micotoxins. Aflatoxin mainly affects cereals and oil seeds during growth and post harvest period due to improper storage of food. *Claviceps purpurea* causes ergotism

Parasites

Parasites get into food in many ways. Some grow in gastro intestinal tract of man and animals and others are naturally occurring in the tissues of animal foods. Health problems arising by these parasites are common in the developing countries particularly in India. *Entamoeba histolytica* causes amoebic dysentery and the major cause is faecal contamination. WHO reports that deaths due to amoebiasis is higher as compared to other parasitic infections and approximately 100,00 deaths occur every

year around the world. World widely about 1200 million people are infected with *Ascaris lumbricoides*. This is also from faecal contamination. *Ancylostoma duodenale* commonly called as hookworm may reach the upper intestine through blood stream and may cause anaemia.

Food borne diseases caused by some pathogenic organisms

Pathogenic organisms	Food community involved	Ill effects and diseases
1) BACTERIAL		
<i>Bacillus cereus</i>	Cereal products	Nausea, vomiting, abdominal pain
<i>Clostridium botulinum</i>	Defectively processed meat & fish	Botulism (muscular), paralysis, death due to respiratory failures
<i>Clostridium perfringens</i>	Defectively processed precooked meat	Nausea, abdominal pain and diarrhea
<i>Salmonella</i>	Defectively processed meat, fish & egg products, raw vegetables grown on sewage	Salmonellosis (Vomiting, diarrhea and fever)
<i>Shigella sonnei</i>	Foods kept exposed sale in unhygienic surroundings	Increased salivation, vomiting, abdominal pain and diarrhea
<i>Streptococcus pyrogenes</i>	Foods kept exposed sale in unhygienic surroundings	Scarlet fever, septic sore throat
Pathogenic organisms	Food community involved	Ill effects and diseases
2) FUNGAL		
<i>Aspergillus flavus</i> (Aflatoxin)	Corn and groundnut	Liver damage and cancer
<i>Claviceps purpurea</i>	Rye and pearl millet infested with ergot	Ergotism (burning sensation in extremities)
<i>Fusarium trichothies</i>	Cereals and millets infected with fusarium	Alimentary toxic

<i>trichiodies</i>	fusarium	
<i>Penicillium islandium</i>	Rice	Liver damage
3) PARASITIC		
<i>Trichinella spiralis</i>	Pork and pork products	Nausea, vomiting, diarrhea, colic and muscular pains (trichionosis)
<i>Ascaris lumbricoides</i>	Raw vegetables grown on sewage farms	Ascariasis
<i>Entamoeba histolytica</i>	Raw vegetables grown on sewage farms	Amoebic dysentery
<i>Ancylostoma duodenale</i>	Raw vegetables grown on sewage farms	Epigastric pain, loss of blood, anaemia

Measure to prevent microbiological hazards

- ❖ Avoid contamination from raw material, ingredients and process aids
- ❖ Prevent contamination from transport vehicles entering the premises
- ❖ Keep raw material, packaging material and food ingredients protected from contamination
- ❖ Maintain proper hygiene in the processing area and immediate vicinity outside the plant
- ❖ Control traffic movement in high hygiene areas
- ❖ Avoid contamination from utensils, and food contact surfaces
- ❖ Train on importance of hygiene and control personal hygiene and habits

CHEMICAL HAZARDS

Chemicals are being contaminated in our food in a number of ways. Chemical contaminants are wide spread in our environment and may enter the food chain and be present in all plant and animal products we eat (eg dioxins). Chemical components of materials which come in to contact with food such as packaging materials may be

absorbed to our foods. Chemical components may form during food processing or cooking. (e.g. polycyclic aromatic hydrocarbons). To increase the food production, use of synthetic fertilizers, pesticides, and herbicides became inevitable (Rao, 2002). Chemicals used in farming such as pesticides and veterinary medicines may remain in products we eat. Additives are deliberately added to food in order to provide some useful purpose such as flavours and preservatives. They can also have some toxic effects (eg. Aginomotto).

Some natural components of plants may themselves cause toxicity (glycoalkaloids in potatoes). While some may be harmful if not cooked properly (eg lectins in pulses). There are also some foodstuffs, which can cause allergies in susceptible individuals. Chemicals may be produced by moulds, which contaminate crops during storage such as aflatoxins.

Human exposure to chemicals and nutritional imbalances are currently known or suspected to be responsible for promoting or causing cancer, kidney and liver dysfunction, hormonal imbalances, immune system suppression, musculoskeletal disease, birth defects, premature births, impeded nervous and sensory system development, reproductive disorders, mental health problems, cardiovascular diseases, genital urinary diseases, old age dementia, and learning disabilities (Babu and Rao, 1996)

Toxic effects of some metals & chemicals

Name	Foods commonly involved	Toxic effects
1) Arsenic	Fruits sprayed by lead arsenate	Dizziness, chills, cramps, paralysis leading to death
2) Barium	Foods contaminated by barium carbonate	Violent persistent muscular twitching and convulsions
3) Cadmium	Fruit juices and soft drinks that come in contact with cadmium and plated vessels	Excessive salivation, liver, kidney dysfunction, prostate cancer, multiple fractures (Painful 'Itai-Itai' disease reported from Japan due to Cadmium poisoning)

4) Cobalt	Water, Beer	Cardiac failure
5) Copper	Acid foods in contact with tarnished copper ware	Vomiting diarrhoea, abdominal pain
6) Lead	Some processed foods, lead water pipes	Paralysis, brain damage
7) Mercury	Mercury contaminated fish, fungicides	Paralysis, brain damage & blindness
8) Tin	Canned foods	Colic, vomiting, photophobia
9) Zinc	Foods stored in galvanized iron ware	Dizziness, vomiting

Food Additives

Food additives are defined as substances added intentionally to food, generally in small quantities, to improve its appearance, flavour, texture or storage properties (WHO, 1987)

Types of food additives

- Colours
- Enzymes
- Texturisers
- Antioxidants
- Sequestrants
- Anticaking agents
- Leavening agents
- Sweetening agents
- Preservatives
- Emulsifiers and stabilizers
- Humectants
- Flavouring agents
- Buffering agents
- Nutritive additives

- Flour improvers
- Acidulants
- Fat replacers

Food and Drug Administration has approved many compounds as safe for addition to food in prescribed levels. These are generally referred to as Generally Recognized as Safe Substances (GRASS).

Food Colourants

Synthetic food colourants and their acceptable daily intake

	Colour	Name	ADI (mg/kg body weight)
A	Red	1. Amaranth	0.75
		2. Carmosine	1.25
		3. Erythrosine	1.25
		4. Ponceur 4R	0.125
B	Yellow	5. Sunset yellow	2.5
		6. Tartrazine	7.5
C	Green	7. Green S	5.0
		8. Fast green	12.5
D	Blue	9. Brilliant blue	2.5
		10. Indigo carmine	5.0

(Swaminathan, 1997)

Synthetic food colourants above safe limits causes asthma, allergy, hyper activity, thyroidism, and degenerative changes in stomach, liver, kidneys and abdomen. In India, various surveys indicated that permitted colours are used even 730ppm when the permitted level is only 100 ppm in foods. Metanil yellow, causes insufficient oxygen supply to skin, and mucus membrane along with degenerative changes in the stomach, liver, kidneys, and abdomen. Intake of lead chromate results in epigastric pain, anaemia, nausea, and constipation. Consumption of rhodamine lead to growth retardation, and haemolysis of blood cells, while sudan dyes produce kidney lesions (WHO, 1987).

Texturisers

Urea is used in the food industry as a texturiser in chewing gum, synthetic milk, and alcoholic beverage. Urea causes some toxic effects like hepatic disorders, neuropsychiatric disorders, carcinogenicity and mutagenicity (Babu and Bhat, 1996).

Antioxidants

Antioxidants are the substances when added to food will retard or prevent the oxidative deterioration of foods.

Permissible limit of permitted antioxidants in foods

Food	BHA	BHT	PG	Total allowed
Fried foods and baked products	200	200	200	200
Dry breakfast cereals	50	50	-	50
Unsmoked dried sausages	30	-	-	30
Chewing gum	1000	1000	1000	1000
Active dry yeast	1000	-	-	1000

Sequestrants

Permissible limits of sequestrants in foods (ppm)

Sl. No.	Food stuff	Sequestrants	Permitted level
1	Fats and oils	Citric acid	100
2	Salad dressing & spreads containing emulsified fats	Na EDTA	75
		NA citrate	100
3	Roasted nuts & nut meat	Citric acid	100
4	Fried and baked foods	Citric acid	100
		EDTA	75

Anticaking agents

Prevents caking by moisture absorption. Eg. Carbonates and Phosphates of Ca and Mg. Maximum permissible limits - 2 per cent.

Emulsifiers

They are the Surface active agents which help to disperse or emulsify two immiscible liquids to give a stable mixture. Eg. Mono and diglycerides, and BVO, which is banned in India. Safe level - 0.2 per cent in bakery products and chocolates.

Sweetening agents

Used as a substitute for cane sugar. Eg. Sodium salt of saccharin, aspartame, sorbitol, mannitol etc. Saccharin induces bladder cancers, hyper activity, anorexia, vomiting, diarrhea, abdominal pain etc. Safe level - 15-mg/kg body weight. FDA has banned the use of cyclamates since it has found to be causing bladder cancer.

Preservatives

Any substance, which is capable of inhibiting, retarding or arresting the growth of microorganism is termed as preservative. According to FPO, the maximum amount of sulpherdioxide, allowed in squash, crush and cordial is 350 ppm and in RTS and nectar 70 ppm

Benzoic acid- according to FPO, permitted level in RTS and Nectar is 120 ppm and in squash, crush and cordial is 600 ppm

Sorbic acid-Permitted levels -0.1-0.4%

Propionic acid -permissible levels are 0.1-0.4%

Nitrites and nitrates- permitted levels are 50-125mg/kg

Nitrate is used in meat products in level of 150-500 mg/kg food

Sulpherdioxide, sulphites and metabisulphites have been found to precipitate allergic reactions in about 10 % of asthmatic patients the symptoms of adverse effects of sulpherdioxide may manifest dizziness, chest pain, eye irritation and difficulty in breathing (Aruna et al, 1997).

According to FPO, Food is considered adulterated when-:

1. Admixture of inferior and cheaper substance
2. Extraction of quality ingredients from the food
3. Preparing or packing under unsanitary conditions

4. Sale of insect infested food
5. Obtaining food from a diseased animal
6. Incorporation of poisonous component
7. Entry of injurious substance from container used
8. Use of additives above safe limits
9. Sale of substandard product, which may or may not be injurious to health

Situations In Which Food Additives Are To Be Avoided-:

- In baby foods
- To mask spoilage or bad quality of food and thus deceive the consumer
- To cover up defects in handling and processing
- To make food attractive and appealing to consumer at the expense of its nutritional quality and safety
- To obtain a desired effect instead of adopting improved processing techniques
- Addition adversely affect health of the consumer

Pesticide residues in food

Pesticide residue means any specified substance in food, agricultural commodities or animal feed resulting from the use of pesticide. The term includes any derivatives of pesticide such as conversion products, metabolites, reaction products, and impurities considered to be of toxicological significance.

The role of agrochemicals in modern agriculture is continuously evolving. Pesticide residues can remain even when pesticides are applied in the right and at the right time. Several human and animal health effects, including cancer, hormonal and reproductive disruption, decreased immune function, chemical sensitivity and neurotoxicity have been associated with chemical pesticides.

Pesticides and Health Implications

The chief constituents of agrochemicals are inorganic fertilizer, which supply the major plant nutrients and numerous plant protection chemicals, insecticides, fungicides, herbicides. The major cause of pesticide contamination is their poor sensitivity over use, unsafe and improper methods of application. Studies on pesticide contamination of food in India reveals that our food is becoming toxic and crossing the threshold level fixed by the WHO. An average daily Indian meal contains half a

milligram of DDT and BHC and this is 44 times more than an average American meal. In a study of farm workers who sprayed DDT and malathion regularly it was found that 50 percent of workers developed system of anxiety, sleep disturbances, depression, and headache, blurred vision and impaired memory (prakash, 2003).

Food stuffs collected from different regions of India were analysed for the presence of HCH (BHC), DDT, HCB, aldrin, dieldrin, heptachlor, and PCBs. Significantly high levels of food contamination with HCH, DDT, aldrin and dieldrin were evident through out India. The average daily intake of HCH and DDT by Indians were estimated to be 115 and 48 mug/person, respectively which were higher than those observed in the most of the developed nations (Kannan, *et.al.* 1998)

In Karnataka, since 1975, over 300 people have been struck by a mysterious crippling attack of arthritis called "Handigodu syndrome". The organophosphorous block acetyl choline in the nervous system and caused dizziness, nausea, head ache, diarrhoea, numbness, and visual disturbances. These compounds may cause foetal abnormalities and are carcinogenic (Rao, 2002)

Dube (1995) reported that 426 pesticides tested on animals 40% produced skin irritation, carcinogenic tumor and mutagenic damage

In a nutshell, the health hazards by the misuse or indiscriminate use of pesticides leads to-

1. Enzyme imbalance
2. Skin and allergic reactions
3. Delayed neuro sensitivity
4. Behavioral changes
5. Effect on reproductive system
6. Respiratory failure
7. Effects on immune system
8. Cataract formation
9. Loss of memory
10. Carcinogenic changes

(Babu and Rao, 1996)

Presence of Veterinary Drugs in Foods

Veterinary drug means any substance applied or administered to any food-producing animal, such as meat or milk-producing animals, poultry, fish or bees, whether used for therapeutic, prophylactic or diagnostic purposes or for modification of physiological functions or behaviour

Anti microbial veterinary drugs are mostly used for treatment purposes, prophylactic purposes and also as feed additives to promote growth, enhance feed acceptability, improve feed efficiency and breeding efficiency. The indiscriminate use of these drugs has led to occurrence of residues in various food products of animal origin. Drugs most commonly involved in residues include streptomycin, penicillin, sulfamethazine and oxytetracycline in a study conducted by FDA.

Residues of drugs in foods are found to have many public health risks

- A) Antibiotics such as penicilins and cephalosporins have the potential for acute allergic reactions
- b) Chloramphenicol is associated with aplasia of bone marrow
- c) Emergence of antibiotic resistant bacteria in animals and transfer of resistant genes to human pathogens
- d) They are found to interfere with human gut microflora
- e) Compounds such as nitrofurans have been found to be carcinogens
- f) Tetracyclins and its derivatives causes immuno depression, phyto toxicity
- g) Sulphonamides may induce thyroid adenoma and hyperplasia in lad animals (Lee *et. al*, 2001)

Measures to prevent chemical hazards

- ❖ Use only approved chemicals of acceptable purity
- ❖ Have detailed product specifications for chemicals entering plant
- ❖ Maintain letters of guarantee from suppliers especially for food grade ingredients and packing materials
- ❖ Inspect vehicles / containers used to ship finished product
- ❖ Properly label and store all chemicals
- ❖ Use and store pest control chemicals with due care

- ❖ Properly train employees who handle chemicals.

FOOD QUALITY AND CONTROL MEASURES

Quality of foods can be defined as the composite of those characteristics that differentiate individual units of a product and have significance in determining the degree of acceptability of that unit by the buyer (Premavalli, 2000). Food quality can be considered as a complex characteristic of food that determines its value or acceptability to consumers. Besides safety, quality attributes include: nutritional value; organoleptic properties such as appearance, colour, texture, taste; and functional properties.

In our country quality standardization systems have come in to force for regulating food manufacturing operations. Two categories of standardization systems compulsory legislations and voluntary standards are formulated in India.

STANDARDIZATION SYSTEMS IN INDIA

The laws are governed by numerous legislations and implemented by various ministries. Indian food laws are given below

Act / Order	Mode of operation	Regulations	Special features
I. Compulsory legislation: I. Prevention of Food Adulteration Act [1954(PFA Act)]	Ministry of Health & Family Welfare Directorate General of Health Services Central Committee for Food Standards	Makes provision for prevention of adulteration of food Adulterated, misbranded, not in accordance with the conditions of License shall be prohibited for selling Proprietary foods shall specify the ingredients in the product in the descending order of their composition of the label	Minimum quality standard Ensures safety against harmful impurities, adulteration. Mandatory. Non-following of PFA Act leads to fine and imprisonment

<p>IA. Atomic Energy Rules 1991 (Control of irradiation of food)</p> <p>IB. The infant milk substitutes, feeding bottles and infant foods (Regulations, production, supply and distribution) Act, 1992</p>	<p>Dept. of Atomic Energy</p> <p>Ministry of Health & Family Welfare Directorate General of Health Services Central Committee for Food Standards</p>	<p>Regulates the irradiation application in foods. Certification with the dose & purpose is insisted upon.</p> <p>Regulate the production, supply and distribution of infant products</p>	<p>Certificate of irradiation indicating the dose & the purpose shall be provided by the competent authority.</p> <p>Rules to exercise the powers of Act, 1992</p>
<p>2 Essential Commodity Act 1954</p> <p>2.1 Fruit Products Order (FPO) 1955</p>	<p>Ministry of Food</p> <p>Ministry of Food Processing Industry Central Fruit Products Advisory Committee</p> <p>Ministry of Civil</p>	<p>Regulate the commodities manufacture, commerce and distribution</p> <p>Regulates the manufacture and distribution of all fruits and vegetable products</p> <p>Licence shall be issued after the satisfaction of quality of product, sanitation, personnel, machinery, equipment and work area requirements as per the schedule specified</p>	<p>Formulations of other suborders for easy implementation</p> <p>Licensing authority 'FPO' standard mark shall be imprinted on the products</p>

<p>2.2 Solvent Extracted oils, Deoiled meal and Edible Flour Control Order, 1967</p>	<p>Supplies</p>	<p>Regulates the production and distribution of solvent extracted oil, deoiled meal, edible flours. Specifications of the products provided.</p>	
<p>2.3 Vegetable oil products (Regulation) Order, 1998</p>	<p>Ministry of Food and Consumer Affairs</p>	<p>Regulates the production and distribution of all the edible oils. Specifications of the products provided.</p>	<p>Supersedes the vegetable oil products (Control) Order, 1947 and vegetable oil products (Standards of quality) Order, 1975.</p>
<p>2.4 Meat Food Products Order, 1975</p>	<p>Ministry of Food & Civil Supplies Directorate of Marketing & Inspection</p>	<p>Regulate the manufacture, quality and sale of all meat products.</p>	
<p>2.5 Sugar (Control) Order, 1966</p>	<p>Ministry of Agriculture and Irrigation Department of Sugar</p>	<p>Regulates the manufacture, quality and sale of sugar</p>	
<p>2.6 Export (Quality Control & Inspection) Act, 1963</p>	<p>Ministry of Commerce Export Inspection Council 5 Regional Export Inspection Agencies Network of 50 Offices</p>	<p>Regulates compulsory, pre- shipment inspection Exportable Commodities list has been notified for pre- shipment inspection. Quality control of various export products is monitored</p>	<p>AGMARK has been recognized as an agent for inspection and quality control of certain items.</p>

<p>2.7 Standards on Weights and Measures Act 1976</p>	<p>Ministry of Food & Civil Supplies Directorate of Weight and Measures</p>	<p>Prescribed the conditions for packed products with respect to quantity declaration, manufacturing date and sale price.</p>	<p>Protecting the consumer in general by guaranteeing the quantity for the amount paid.</p>
<p>2.8 The Consumer Protection Act 1986</p>	<p>Ministry of Food and Civil Supplies</p>	<p>Provision made for the establishment of consumer councils and other authorities for the settlement of consumer disputes.</p>	<p>Protection of the interest of consumer.</p>
<p>II. Voluntary Standards</p>			
<p>1. Agricultural Produce (Grading & Marketing) Act, 1937</p>	<p>Directorate of Marketing and Inspection</p>	<p>Prescribed the conditions for Grade Standards are prescribed for Agricultural and Allied Commodities Grading, sorting as per quality attributes and inspection are included</p>	<p>AGMARK Certificate System</p>
<p>2 Bureau of Indian Standards</p>	<p>Indian Standards Institution</p>	<p>Prescribing of Grade Standards, Formulation of Standard Specification of foods, Prescribing standards for limits of toxic compounds as applicable</p>	<p>General cover on hygienic conditions of manufacture, raw material quality and safety are given Quality & Safety oriented standard Enforces certification system.</p>

INTEGRATED FOOD LAW

The latest report by the industry where by it is proposed to converge all the laws relating to food quality and safety in a single integrated food law (Chanda, D.S., 2004). It has been provided to government and group of ministries constituted by government. Integrated food laws calls for the constitution of

1. Food Regulatory Authority of India (FRAI)

To set up rules and working guide lines to defeat standards to notify list of registered independent certified agencies, to notify accredited laboratories encourage adoption of food quality assurance system i.e., GHP, GMP, HACCP etc.

2. Council of Food Standards

To be constituted under a chairman of eminence in the field of food science and technology with a total of 11-15 members of the council such a council to formulate the minimum food safety standards essential compositional standards, for selected food items and guidelines labeling

3. Food Standards Administration System (FSA).

The states under the overall supervision of the central government will do this

INTERNATIONAL FOOD STANDARDS

Two organizations namely, International Standards Organisation (ISO), and Codex Alimentarius Commission (CAC) are working at the international level

Codex Alimentarius Commission (CAC)

The term codex alimentarius is taken from latin and means food code. Codex alimentarius commission was established to develop agreements on international standards and safety practices for foods and agricultural products. The codex sets the minimum quality, safety and hygienic standards that countries voluntarily adhere to

importing and exporting food products. The codex contract points in India is the Directorate General of Health Services (DGHS) in the ministry of health however ministry of food processing industries is closely associated with the activities of codex alimentarius.

Besides, standards belonging to a common subject group have been grouped and are available in separate volumes of codex alimentarius volumes contain general principles, general standards, definitions, codes, commodity standards, methods and recommendations.

HACCP-Hazard Analysis And Critical Control Point

HACCP is a food safety management system, is a science based system which systematically identifies specific hazards and provides measures for their control to ensure safety of food (Soharb, 1999).

HACCP is an efficient and preventive quality assurance method providing not only food safety, but also minimization of waste and pollution of environment. Even though many developed countries are enforcing HACCP as mandatory requirement of food production and processing, Indian union and state government are yet to introduce HACCP as an essential criteria for our food processing industries as well as a hotel industry.

Hazard analysis and critical control point has become increasingly important for all food business as an effective means of ensuring food safety and as a means of complying with new legislation. This approach is based upon the recommendations of the WHO and FPO and the codex alimentarius commission. The HACCP concept was developed in the 1960's as a system to ensure the safety of food products. The HACCP can be defined as a system, which identifies, evaluate and control hazards, which are significant for food safety. HACCP principles apply to microbiological, chemical and physical hazards associated with foods but are most widely applied to microbiological hazard because they are leading cause of food borne diseases

(Bhat *et al.* 2000).

HACCP have developed a series of seven principles on steps

1. Hazards associated with growing, harvesting, raw materials, ingredients, processing, manufacturing, distribution, marketing, preparation and consumption of a given food are each assessed in detail. Different areas of contamination are measured. This includes both incoming ingredients as well as finished product.
2. Critical control points for controlling each hazard identified above are determined. Control of these points must be maintained in order to ensure the safety of the products.
3. After each critical control point is identified, the limits on that point must defined
4. Specific procedures for monitoring the control point must established
5. The next step is establishing an action plan for taking corrective action when monitoring indicates that the critical control points limits have been exceeded.
6. It is important that the entire HACCP plan be thoroughly documented. It involves keeping of all records of measurements of all critical control points
7. Finally, it is important to have procedures for verifying that the HACCP plan is being followed and it is working according to plan

HAZARDS ANALYSIS AND CRITICAL CONTROL POINTS

RISK ASSESSMENT	HAZARD ANALYSIS	HAZARD MANAGEMENT	
HAZARD IDENTIFICATION	HAZARD IDENTIFICATION	MONITORING	
HAZARD CHARACTERIZATION	ESTABLISHING	CCP'S ESTABLISH	
EXPOSURE ASSESSMENT	CRITICAL CONTROL POINT	CORRECTIVE ACTION	
RISK CHARACTERIZATION	ESTABLISHING CRITICAL LIMITS	VERIFICATION OF SYSTEM DOCUMENTATION AND RECORDS	SAFE FOOD

Pre-requisites of HACCP

The GMP and SSOP are known as the corner stones of food safety and quality and are two of the prerequisite programs required for successful implantations of HACCP plan.

1. Good manufacturing practices (GMP)

Any food processes under unsanitary conditions is considered adulterated because the food may be contaminated with filth or substances that could render the food injurious to health. The current good manufacturing practices and regulations were issued to help to prevent these problems

GMP includes

Facilities-the establishment should be located constructed and maintained according to sanitary design principles. There should be linear product flow and traffic control to minimize cross contamination from raw to cooked materials

Personnel- the plant management should take all reasonable measures and precautions to ensure Disease control, cleanliness, education and training of personnel employed in the organization

Sanitary facilities and control-This includes general maintenance of facilities, cleaning and sanitation of surfaces, pest control etc. plant shall be equipped with adequate sanitary facilities and accommodations. The plant should have adequate supply of water, good plumbing, efficient sewage, disposal toilet facilities, hand washing facilities etc

Equipment and utensils-This include the design, specification and cleaning methods of all equipments and utensils installed for production.

Process control-All operations including receiving, inspecting, transporting, segregating, preparing, manufacturing, packaging and storing of food should be conducted in such a way that it does not lead to contamination.

Personal hygiene-All employees and other persons who enter the manufacturing plant should follow the requirement for personal hygiene.

Training-All employees should receive documented training in personal hygiene, GMP, cleaning and sanitation procedures, personal safety and their role in the HACCP program.

Chemical control-Documented procedures must be in place to assure the segregation and proper use of non-food chemicals in plants. These include cleaning chemicals, fumigants and pesticides or baits used in or around the plant.

Trace ability and recall - all raw materials and products should be lot coded. A recall system should be in place so that rapid and complete traces and recalls can be done when product retrieval is necessary.

Pest control-Effective pest control programmes should be in place.

Sanitation standard operating procedures (SSOP)

SSOP develop the basis for maintaining a food facility that has a clean, sanitary environment that is free of food hazards.

Benefits of HACCP

- ❖ Reduces contamination
- ❖ Reduces product destruction
- ❖ Provides market protection
- ❖ Provides preferred supplier status
- ❖ Demonstrated conformance to international standards and regulations requirements and overseas markets
- ❖ Transforms commodities into branded products
- ❖ International acceptance

(Sarkar *et al.*, 2005)

ISO900 series standards

It has found in 1946 ISO consist of approximately 90 member countries at present ISO is responsible for the promotion and development of international standards and related activities, including conformity assessment such as testing, inspection, laboratory, accreditation, certification and quality assessments ISO9000series standards provide the requirement to which organization desirous of certification must conform. One of the important aspects of the ISO registration process is to verify whether the unit seeking registration is indeed doing what is being claimed in its quality manual. The process should begin with familiarization with the standards followed by an assessment of the current quality assurance system.

ISO certification is a base line model for quality assurance. Concerning a new approach towards technical harmonization and standardization, the European committee has adopted 8 modules. Companies exporting any of the products covered under the directives will need to conform to the requirements of applicable module

- a) Manufacturer's self declaration of conformity
- b) EC type examination
- c) EC declaration of conformity to type
- d) Production quality assurance
- e) Final inspection and testing
- f) Product verification
- g) Unit verification by EC

If a company is exporting to the European Union, a product covered under one of the directives, which require conformity with the above modules. ISO technical committee 176 published in 1987 and updated approximately 5 documents whose focus is quality assurance systems.

National food control system suffer serious inadequacies including

- They are not based on modern scientific management concepts using compliance policies, risk assessment, HACCP, etc.
- In sufficient involvement of scientific expertise from the academia, industry, consumers to strengthen the scientific basis for food control decision making processes
- Lack of suitable facilities such as laboratories
- Lack of resources such as trained inspectorate and laboratory staff, funding
- Inflexibility of the system making it difficult to cope with developments in food science and technology, changing consumer demands, and newer requirements of trade and industry
- Lack of coherence among different governmental activities concerning agriculture, food, trade, industry and health

Conclusion

All most all countries have food control system to protect their populations against unsafe adulterated or otherwise poor quality food. In developing countries large quantities of food pass through a multitude of food handlers and middleman extending the food production, processing storage and distribution chain, control is more difficult and there is greater risk of food contamination and adulteration. Access to good quality food is a basic right that should be ensured by the nation. Ensuring safe food with out contamination, absence of toxins and pathogens, to the consumers is a global challenge, which requires in depth technology and research.

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Discussion

1. What is organic food? What are the advantages of organic foods?

Ans: Organic is a labelling term that denotes products that have been produced in accordance with organic production standards and certified by a duly constituted certification body or authority. Organic agriculture is based on, avoiding the use of synthetic fertilizers and pesticides.

It ensures safe food with zero contamination

2. Which is the most common type of contamination in foods?

Ans: Biological contamination

3. What are the health hazards caused by the usage of Aluminum foils in food packaging?

Ans: Aluminium can leach out in to food from foils and containers made out of aluminium. Aluminium toxicity can cause demensia, osteodystrophy, and Alzheimer's disease

4. Is there any quality standards for Aurevedic drugs?

Ans: There is no quality specifications for aurevedic drugs and health care products

5. Which all industries follow HACCP regulations?

Ans: All leading sea food industries follows HACCP. Major food exporting companies are also follows this regulation

6. Is there any specified standards for mineral water?

Ans: Mineral water is provided with ISI standards

7. What is mean by leavening agent?

Ans: leavening agents are used to incorporate carbon dioxide in dough, batter etc. Eg yeast

8. What is your opinion about the future perspective of food safety and quality?

Ans: International quality standards and specifications are now implemented mainly in export oriented industries. But the local market is still in very pathetic situation. Implementation of quality standards from the very grass root level, only ensure food safety to all. Then only the nation's health status will improve

9. What are the quality standards for GM foods in India?

Ans: Not specified yet in India

10. Are there any standards in India, which are followed by exporters and importers?

Ans: All manufacturers of processed foods follows the quality standards and specifications, recommended by Indian food laws.

11. How a consumer knows whether the manufacturer is having HACCP?

Ans: By examining the logo of HACCP.

12. Name a disease recently came in to news that caused by the consumption of beef meat?

Ans: Anthrax

ABSTRACT

Access to good quality food has been man's endeavour from the earliest days of human existence. Safety of food is the basic requirement of food quality. Food safety is the assurance that damage or injury from the use of a food substance is impossible. Food systems in developing countries are not always well organized and developed as in the industrial world. Therefore people in developing countries are exposed to a wide range of potential food quality and safety risks. Producing good quality and safe food is a pre-requisite to successful and sustainable development of national agricultural resources, domestic and international food trade (Prakash, 2003).

Consumption of unsafe, contaminated food leads to food borne diseases, which cause considerable morbidity and mortality (Raghuramaiah, 2001). To increase the food production, use of synthetic fertilizers, pesticides, and herbicides became inevitable. The use and abuse of these synthetic chemicals results in chemical contamination of food, which is a serious public health problem (Rao, 2002).

Quality of foods can be defined as the composite of those characteristics that differentiate individual units of a product and have significance in determining the degree of acceptability of that unit by the buyer (Premavalli, 2000). In our country, quality standardization systems have come into force for regulating food manufacturing operations. Two categories of standardization systems, compulsory legislations and voluntary standards are formulated in India. Two organizations viz. International Standards Organisation (ISO), and Codex Alimentarius Commission (CAC) are working at international level. The Codex General Principles of Food Hygiene has recommended a Hazard Analysis Critical Control Point (HACCP) based approach as a means to enhance food safety (Sohrab, 1999). Ensuring safe food without contamination, absence of toxins, and pathogens to the consumers is a global challenge, which requires in depth technology and research.

DIET AND DIABETES

By

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(2004-24-02)**

**Ph.D Home Science
(Food Science and Nutrition)**

SEMINAR REPORT

**Submitted in partial fulfillment for the requirement of the
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**DEPARTMENT OF HOME SCIENCE
COLLEGE OF HORTICULTURE
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2005**

DECLARATION

I, Sharon,C.L.(2004-24-02) hereby declare that the seminar entitled "Diet and Diabetes" have been prepared by me, after going through the various references cited here and has not been copied from my fellow students.

Vellanikkara



Sharon,C L
(2004-24-02)

CERTIFICATE

This is to certify that the seminar report titled “DIET AND DIABETES” has been solely prepared by Sharon, C.L (2004 – 24 – 02), under my guidance, and has not been copied from any seniors, juniors or fellow student’s seminar reports.

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CONTENTS

Sl.No.	Title	Page No.
1.	Introduction	1
2.	Prevalence	1
3.	What is diabetes	2
4.	Definition	2
5	Types	4
6	Diagnosis	10
7	Aetiology/Risk factors of diabetes	15
8	Metabolism in diabetes	19
9	Complications	19
10	Treatment	26
11	Diabetic diet prescription	30
12	Dietary guidelines	30
13	Conclusion	48
14	References	50
15	Discussion	53
16	Abstract	

DIET AND DIABETES

1. INTRODUCTION

Diabetes mellitus or 'Madhumeham' has been known for centuries as the disease related to sweetness. Persons with diabetes have sugar in their blood and urine. However there is no need to worry, as diabetes can be kept under control with certain changes in the life style -food intake, exercise and the regular intake of the prescribed medicines. Even though more than 30 million people all over the world is affected with diabetes not all are well informed about the nature of their disease.

2. PREVALENCE

Diabetes mellitus is a heterogeneous disorder of multifactorial origin of which many sub types are present. There is every reason to suppose that diabetes will remain a threat to public health in this 2000 era and beyond. Demographic and epidemiological evidence suggests that in the absence of effective intervention diabetes will continue to increase in frequency, worldwide. According to WHO, the number of people with diabetes throughout the world is expected to double by 2030 (Wild, 2004)

Many developing countries including India are facing major challenge of adult morbidity and mortality due to various diseases with particular reference to diabetes mellitus. Diabetes is one of the leading causes of death and it ranks 3rd among chronic diseases (Rema *et al.*, 1997). Diabetes in India will increase by 59% compared to 41% in other Asian countries by 2025 (Agarwal, 2001). Indians have the highest prevalence of diabetes among the Asian countries (ADA, 2003)

Surveys showed that diabetes was as common in urban India as among migrant Indians (Ramaiya *et al.*, 1990). India already has the highest number of adult diabetes cases (33 million) and this number is expected to rise to 57 million by 2020 and 57.2 by 2025 (Narayanan *et al.*, 2000). It is believed that diabetes is more prevalent in affluent societies where obesity is a major health problem. However, it has been observed that undernourished subjects are equally susceptible to diabetes.

In India, 2-4% of the population is affected with diabetes, and more than 90% of all diabetics belong to the non-insulin dependent type.

In southern Kerala, the overall crude prevalence of NIDDM is 59% highest in urban 12.4% followed by midland (8.1%), highland (5.8%) and coastal (12.5%) regions (Kutty *et al.*, 2000).

3. WHAT IS DIABETES?

We require energy for our daily activities. This energy is mainly derived from carbohydrates. When we eat carbohydrate in our diet, our blood sugar increases. If it rises beyond 180mg/100 ml, sugar is excreted in the urine. Normally insulin, a hormone produced by the beta- cells of the pancreas help to utilize sugar for the production of energy by the body. It helps in glucose uptake by the cells, prevents a rise in blood sugar and maintains its level within certain normal limits. In diabetes, the pancreas cannot produce enough insulin or what is produced is not effective in controlling the blood sugar, this affects the metabolism of several nutrients, with varying ill-effects.

4. DEFINITION

Diabetes (medically known as Diabetes mellitus) is the name given to disorders in which the body has trouble regulating its own blood glucose or blood sugar levels (Jerreat, 1999)

Diabetes mellitus is a chronic metabolic disorder that prevents the body to utilize glucose completely or partially. It is characterized by raised blood glucose concentration in the blood and alterations in the Carbohydrates, protein and fat metabolism (Sreelakshmy, 2000)

Diabetes mellitus (diabetes = flow through, mel - honey) is a chronic metabolic disorder with strong hereditary bases, associated with high blood sugar and usually with passage of sugar in the urine (Antia and Abraham, 2000)

5. TYPES (CLASSIFICATION)

The new classification encompasses both clinical stages and etiological types of diabetes mellitus and other categories of hyperglycaemia. It contains stages, which reflect the various degrees of hyperglycaemia in individual subjects with any of the disease processes, which may lead to diabetes mellitus.

All subjects with diabetes mellitus can be categorized according to clinical stages, and this is achievable in all circumstances. The stage of glycaemia may change over time depending on the extent of the underlying disease processes. The disease process may be present but not have progressed far enough to cause hyperglycaemia. The aetiological classification reflects the fact that the defect or process, which may lead to diabetes, may be identifiable at any stage in the development of diabetes even at the stage of normoglycaemia.

The most widely accepted classification of diabetes mellitus was published by WHO in 1980 (WHO, 1980) and modified form in 1985 (WHO, 1985). According to this there are mainly 5 types:

- 1) Insulin dependent diabetes mellitus (IDDM)
 - a) Ketosis prone
 - b) Ketosis resistant
 - c) With pancreatic calculi
- 2) Non-insulin Dependent Diabetes mellitus (NIDDM)
- 3) Malnutrition related Diabetes mellitus (MRDM)
 - a) Fibrocalculous Diabetes Mellitus (FCDM)
 - b) Protein deficient Diabetes Mellitus (PDDM)
- 4) Impaired glucose tolerance (IGT)
- 5) Gestational Diabetes Mellitus (GDM)

5.1. TYPE I or Insulin Dependent Diabetes Mellitus (IDDM)

In insulin dependent diabetes mellitus, also known as juvenile onset, patients depend on insulin. The onset is usually sudden and occurs in the younger age group (10 – 12 yrs) or during adolescence and there is an inability of pancreas to produce

adequate amount of insulin. This may be caused by viral infection or due to autoimmunity. Viral infections can cause destruction of β cells. The antibodies developed destroy the β cells. Autoimmunity as the body destroys its own β cells because they appeared to be foreign.

The child is usually underweight. Ketoacidosis is common. Due to damage of the β cells of the pancreas, insulin levels are very low. Such individuals require insulin for survival. The symptoms get severe when insulin is discontinued.

This type can be further classified in to three

a. Ketosis-prone

These are especially seen in the west. Usually the diabetes is of sudden onset after a viral infection, with circulating antibodies to islet cells. Patients are prone to lapse in to coma

b. Ketosis-resistant

These are common in the tropics, originally discovered in Jamaica and labeled 'J' type, they are also seen in India

c. With pancreatic calculi

Young diabetes of this type is commonly seen in south India with intermittent abdominal pain. On plain x-ray of the abdomen, stones are seen in the pancreatic duct

Symptoms of type I are easy to identify because they are specific and severe and because they usually start in otherwise healthy children and young adults. The child or adult becomes tired, very thirsty and starts to urinate frequently during the day and night, even though the person eats more, he or she loses weight

Once the attack has destroyed the β cells life depends on replacing natural insulin. Good health depends on how well this is done. Insulin is ideally given to act at the same time and in the same amount as the pancreas normal release of insulin. Hence insulin injections mimic the pancreas type I diabetes turns out to be a very manageable disease

Symptoms of IDDM

- 1) Polydipsia (Increased thirst)
- 2) Polyuria (Frequent urination)
- 3) Polyphagia (Increased hunger)
- 4) Excessive tiredness
- 5) Irritability
- 6) Weight loss
- 7) Abdominal pain
- 8) Dehydration
- 9) Nausea
- 10) Vomiting

Clinical laboratory test data reveal

- 1) Glycosuria
- 2) Hyperglycaemia

As its onset is abrupt, most often the diagnosis of IDDM is made when the patient first time develops ketoacidosis

5.2. TYPE II or Non-insulin Dependent Diabetes Mellitus (NIDDM)

Non-insulin dependent diabetes or the adult onset diabetes is non-insulin dependent form and develops slowly and is usually milder and more stable. Diabetes mellitus type II is a multifactor disease involving genetic predisposition and various environmental factors (Keen *et al.*, 1982)

In NIDDM insulin may be produced by pancreas but the action is impaired. Nearly 90-95% of all patients with diabetes belong to this category. NIDDM has its onset after 40 yrs of age and affects overweight or obese persons, particularly affects normal or undernourished subjects

Unlike in IDDM, the insulin varies in NIDDM. They are either normal or above normal. Therefore, persons with NIDDM rarely develop ketoacidosis except under conditions of starvation, infections, injury, burns and surgery. Marked peripheral insulin resistance and impaired β cell function both contribute to

hyperglycaemia in these patients. Since insulin secretory capacity is present, though diminished, these patients can be managed with oral hypoglycemic agents.

Symptoms of NIDDM

Type II diabetes usually begins gradually and progress slowly. Many people have no signs or symptoms. Symptoms can be so mild that you might not even notice. Symptoms include:

- 1) Polydipsia (Increased thirst)
- 2) Polyuria (Frequent urination)
- 3) Polyphagia (Increased hunger)
- 4) Fatigue
- 5) Blurred vision
- 6) Usually over weight
- 7) In women, vaginal yeast infections or fungal infections under the breast or in the groin
- 8) Severe gum problems
- 9) Itching
- 10) Impotence in men
- 11) Unusual sensations, such as tingling or burning in the extremities
- 12) Ketoacidosis develops rarely except under stressful conditions such as starvation, infections, injury, burns and surgery
- 13) Hyperglycaemia

Difference between IDDM and NIDDM

IDDM	NIDDM
1. Inflammatory reaction in islets	No insulinitis
2. Islet β cells destroyed	β cells function
3. Islet cell antibodies	No islet cell antibodies
4. Not directly inherited	Strongly genetic bias

5.3. Malnutrition Related Diabetes Mellitus (MRDM)

Malnutrition- related diabetes is mainly seen in some tropical countries like India and it occurs in young people between 15-30yrs of age. Generally people with MRDM are lean and under nourished (Sanjeevi *et al.*, 1992). In this type of diabetes, the pancreas fails to produce adequate insulin. As a result these diabetes require insulin. In contrast to type I diabetes these patients generally do not develop ketoacidosis, when insulin injections are discontinued.

MRDM includes two types:

- a Fibrocalculous Pancreatic Diabetes (FCPD)
- b Protein Deficient Diabetes Mellitus (PDDM)

a. Fibrocalculous pancreatic diabetes (FCPD)

As the name implies, fibrosis and calcification of pancreas are observed. As a result of pancreatitis, abdominal pain is common. Exocrine function is also affected. Since this type of diabetes is found to be common in area of cassava and tapioca consumption, these have been implicated in the aetiopathogenesis of FCPD. It is claimed that linamarin, a cyanogenic glycoside, present in cassava or tapioca in association with malnutrition could be responsible for FCPD. However it is observed that FCPD is present in areas such as Madras, where cassava is not a major food. In addition, surprisingly FCPD is not endemic in all cassava consuming areas. Therefore though cassava hypothesis is attractive, it fails to explain such contradictory findings. It is also suggested that in under nutrition deficiency of sulphur containing amino acids and antioxidants such as b carotene, vitamin E, selenium and zinc, free radicals may cause pancreatic injury leading to FCPD.

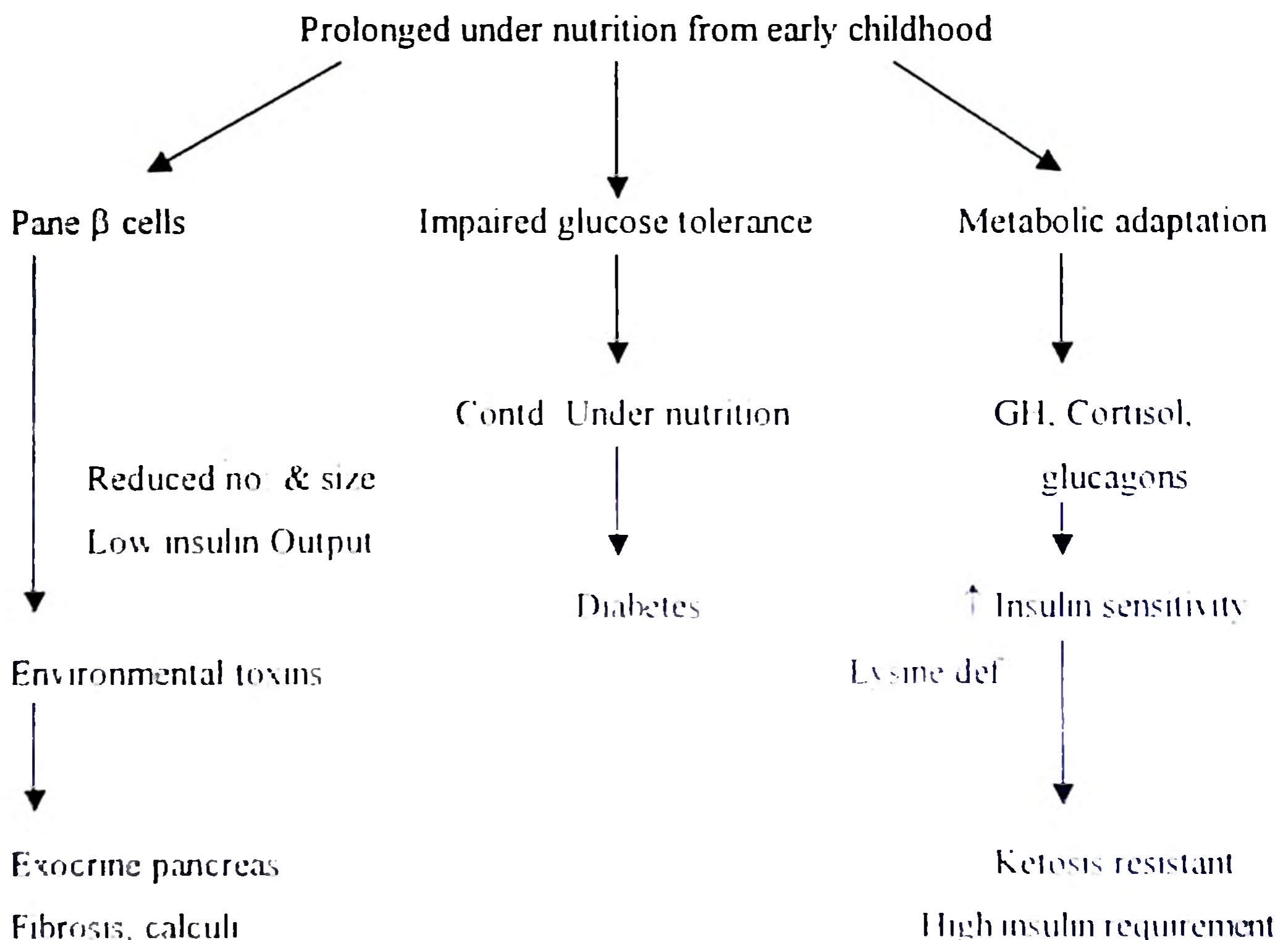
b. Protein Deficient Diabetes Mellitus (PDDM)

Deficiency of proteins and micronutrients without significant involvement of food toxins may produce PDDM. In spite of requiring large quantities of insulin, the

relative resistance of these patients to ketosis have been explained on the basis of presence of residual β cell function and low glucagons levels.

Possible mechanism of diabetogenesis in chronic under nutrition

(Sreelakshmy, 1993)



5.4. Impaired glucose tolerance (IGT)

IGT refers to a metabolic state intermediate between normal glucose homeostasis and diabetes. When the glycaemic response after the administration of a 75 gm of oral glucose load is intermediate between normal and diabetic, the condition is called as IGT. Individuals with IGT are generally free from symptoms of diabetes. However they have the potential to develop diabetes at a later stage. They should be more careful in their diet and avoid obesity. In addition they should undertake regular exercise.

It is estimated that nearly 13-15% persons with IGT develop diabetes in 10yrs with a rate of progression of 1-5% per year. The prevalence of IGT in Indian population in the age group of 30-64 years varies between 7 and 10%.

5.5. Gestational diabetes (GD)

Gestational diabetes is an elevation of blood sugar during pregnancy. Ovarian and placental hormones reduce insulin sensitivity and as a result insulin requirement goes up in pregnancy and if there is no adequate compensating function of β cells, gestational diabetes result.

Diabetes is eliminated to occur in 2-3% of all pregnancies of which 90% are cases of gestational diabetes while rest 10% are established diabetes (Type I or II)(Geol *et al* , 1995)

GD is form of type II diabetes that begins during pregnancy, often near the end of the second trimester or during third trimester. GD increases the diabetes related complications during pregnancy and also is subsequent development of real diabetes after the delivery. There is convincing evidence that mild maternal hyperglycaemia is a risk factor for foetal morbidity, but occurs only in a minority cases. Failure to recognize and treat the condition will result in unnecessary morbidity in some pregnancies, where as overly aggressive approaches to detection and treatment will result in unneeded interventions in many others (Metzyer and Coustan, 1998). Pregnant women who have family history of diabetes or bad obstetric history should be screened for gestational diabetes.

6. DIAGNOSIS

Detection of early diabetes

All adults should have their urine examined for sugar twice a year and more often if

1. Any blood relation has diabetes
2. There is sudden increase in weight
3. There is unexplained weight loss
4. Frequent urination at night

5. Recurrent skin infections
6. Wound doesn't heal
7. Children are over weight at birth

6.1. Random blood sugar

In many cases diabetes can be diagnosed by a single blood estimation, which may be used. A random blood glucose exceeding 200 mg/dl or a fasting blood sugar higher than 120mg/dl is an indication of diabetes.

According to WHO recommendations in a patient with overt symptoms of diabetes, a fasting or random plasma glucose measurement is often sufficient for the diagnosis of diabetes

6.2. Oral glucose tolerance test (OGTT)

The WHO Expert committee on diabetes recommends OGTT as a confirmatory test of diabetes. This test is carried out after 12 hours of over night fasting 75 gm of glucose in adults and 1.75gm/kg body wt in children up to a maximum of 75gm is given orally. All medications should be stopped for atleast 3 days before the test. The fasting blood sample is drawn. Blood sample collected after 2hr after administration of glucose

The diagnostic criteria for diabetes and impaired glucose tolerance test (IGT) are given below

Blood sugar levels (mg/dl)

	IGT	Diabetes
Fasting	< 120	≥ 120
After 75 g glucose load	120-180	≥180

In normal persons without diabetes or IGT, the fasting blood sugar levels vary between 80-110mg/dl. The blood sugar levels increases after the glucose load and come down to basal level within 2 hrs.

6.3. Test for glycosylated hemoglobin (Larren *et al.*, 1990)

Another test examines blood levels glycosylated haemoglobin, also known as haemoglobin A_{1c} (HbA_{1c}). Measuring glycosylated hemoglobin is not currently used for an initial diagnosis, but it may be useful for determining the severity of diabetes. Haemoglobin is a protein molecule found in red blood cells. When glucose binds to it, the haemoglobin becomes modified a process called glycosylation. Glycosylation affects a number of proteins and elevated levels of glycosylated haemoglobin are strongly associated with complications of diabetes. In normal population the glycosylated haemoglobin concentrations vary from 4-7% while in diabetes it is in the range of 8-18% of the total hemoglobin depending on the blood sugar levels.

The test is not affected by food intake, so it can be taken at any time. It reflects the general trend of glucose levels in the blood during the previous 2-3 months.

6.4. Urine examination

Examination of early morning sample of urine may fail to detect early diabetes mellitus. It is necessary to observe the following procedure.

1. Urine is voided just before a meal
2. Breakfast or lunch is taken with the usual helpings of carbohydrate rich foods such as bread, chapattis, rice, fruits and sweets
3. 2 or 3 hrs after meal, urine is voided and examined for sugar
4. Glucose specific dipstick methods are the best
5. Diastix consists of a paper impregnated with enzyme preparation, which give a semi-quantitative estimate of glycosuria (25-3%) when dipped in urine sample containing glucose. Some undoubtedly diabetic people have a negative test owing to a raised renal threshold and non-diabetics with a low renal threshold may give rise to a false positive test.

Ketonuria-the amount of ketone bodies normally excreted by healthy persons is not detected by routine methods. High amounts excreted can be detected in the urine by the nitroprusside reaction, which is conveniently carried out using acetate tablets.

or ketostix, paper sticks. If both ketonuria and glycosuria are found the diagnosis of diabetes is practically certain.

6.5. Benedicts test

Eight drops of urine and 5ml of benedicts solution are taken in a test tube and mixed. The test tube is kept in boiling water for 5 min and color is noted.

Interpretation of Benedicts Test

Appropriate sugar in

Colour	Report	Urine (g%)	Blood (mg%)
Green discolouration	0-trace	-	<200
Green precipitate	+	0.25	200-250
Greenish-yellow precipitate	++	0.5	250-300
Yellowish-orange precipitate	+++	1	300-350
Brick-red precipitate	++++	>2	>350

It is better to carry out this test on the second urine sample collected in the morning. Urinary sugar in this sample will more or less reflect the blood sugar level. Self-monitoring of urinary sugar at frequent intervals provides valuable information to the patient as well as to the doctor in fixing the required dose of drug to control blood sugar. During acute infections or when blood sugar is fluctuating widely during anti diabetic therapy, the urinary sugar must be monitored once or twice daily.

6.6. Glucometer

When testing blood sugars yourself, a glucometer is used. A glucometer is a small device that provides an exact reading of blood glucose. A test strip is used to collect a small drop of blood obtained by pricking the finger with a small specially designed needle (called a lancet). The strip is then placed in the meter. Results are available within 30-45 seconds.

6.7. Glucose sensors

The food and drug administration (FDA) approved the Gluco watch G2 biographer, manufactured by the Cygus Inc, for use in children and adolescents between ages 7 and 17 as a method of detecting trends and tracking patterns in glucose levels.

FDA had approved the original gluco watch for adults use in March 2001. The wrist watch like glucose monitoring devices uses a weak electronic current to draw tiny volumes of tissue fluid through which glucose and other nutrients travel from the bloodstream to the cells, through the skin and then analyze the fluids for glucose levels. As described by the FDA, once the device has been warmed up and calibrated through the use of a finger stick blood glucose test, it is capable of providing up to six painless glucose measurements per hour for 13 hours. The device sounds an alarm if the glucose reaches dangerous levels, alerting patients to a potential problem. The device can store up to 8500 biographical glucose readings over 8 weeks.

Although gluco watch measurements generally are consistent with those of traditional finger stick blood glucose tests, results with gluco watch can differ significantly because these variations are unpredictable. Individual gluco watch readings should never be used to make changes in insulin dose. Gluco watch results should be interpreted with several sequential readings overtime and then confirmed with a finger stick test.

6.8. Medtronic minimed

Another FDA approved, 24hr a day system to measure blood glucose levels, the Medtronic minimed -continuous glucose monitoring system (CGMS), uses a tiny sensor inserted just beneath the skin of the patients abdomen and generally worn by patient for up to 3 days. This device records a glucose measurement every 5 minutes. The user must push buttons on the systems monitor to mark the time of meals, exercise and other pertinent data. After 3 days doctor removes the sensor and downloads the monitor data to a computer for analysis.

7. AETIOLOGY / RISK FACTORS OF DIABETES

Although the actual cause is not clear, the following factors have shown to increase the risk for diabetes

1. Heredity
2. Obesity
3. Stress
4. Infections
5. Diet
6. Age
7. Physical activity
8. Nutritional status

7.1. Heredity

Heredity plays the most important role in conferring susceptibility to diabetes the closer the blood relationship of a person to a diabetic the greater are the chances of developing the disease

Risk of diabetes based on family history

Family history	Risk of diabetes
If both parents are diabetic	99%
One parent diabetic and any other relative of non-diabetic parent is also diabetic	75%
If one parent is diabetic	50%
Any close relative is diabetic	25%

(King *et al.*, 1998)

Between 25-35% of all type II patients have family members with diabetes. Having a first-degree relation with the disease poses a 40% risk of developing

diabetes. People with positive family histories have a higher risk for developing the disease at an earlier stage with more severe features.

Because families share many lifestyles feature (eating and exercise habits) it is difficult to determine whether genetics or environmental play the major role when clusters of diabetes type I and II appear within the families, genetic factor should be strongly suspected.

7.2. Obesity

A strong predisposing factor in middle age diabetes is obesity. Although most type II diabetics are obese, all obese patients do not develop diabetes. Whether or not an obese patient develops diabetes probably depends upon the genetic factors. Increasing weight is a danger signal. Obese people secrete more than the normal amount of insulin with a high glucose load

Obese people in general are less physically active than those whose weight is normal. Middle aged obesity is due to increase in the size of fat cells and with weight reduction, the fat cells and with weight reduction the fat cells decrease in size and the glucose tolerance test may return to normal

It is now recognized that more than obesity parse, the pattern of body fat distribution is important risk factor for diabetes and coronary heart disease. Fat around the abdomen is more dangerous than fat around the hips. Fat deposition is not the same for every individual and is different in men and women. Abdominal android fatness has greater risk.

Android male pattern	Gynoid or female pattern
Upper body obesity Above the waist	Lower body obesity Mainly lower abdomen, buttocks, hips and thighs

Those with upper body obesity have glucose intolerance, which improves with weigh reduction, while those with lower body obesity do not have a tendency to diabetes. Abdominal obesity as assessed by waist/hip ratio (WHR) has been associated with higher prevalence of diabetes in several epidemiological studies.



WHR is an indicator of abdominally located adipose tissue. WHR <1 for man and <0.8 for women.

How obesity leads to Diabetes?

1. ↑ FFA → ↓ Hepatic clearance of insulin → hyperinsulinemia
2. ↑ FFA oxidation by muscles → Insulin resistance (IR)
3. ↑ Secretion of α TNF and leptin by adipose tissue → IR
4. IR → hyperinsulinemia

Increase in body fat, especially visceral fat, mobilizes excess free fatty acids in the portal circulation, which in turn reduces hepatic clearance of insulin, causing peripheral hyperinsulinemia. Utilization of excess FFA's by muscle at the expense of glucose may contribute to the peripheral insulin resistance in obesity. Further increased secretion of α tumour necrosis factor and leptin by adipose tissue in obesity has been involved in insulin resistance (Sumathi, 2000)

7.3. Stress

Stress is a non-specific response of the body to any demand made upon it

There are three types

- a. Acute (unexpected events)
- b. Chronic (bereavements)
- c. Intermittent (conflicts)

Stress causes

- The heart to beat faster
- B P to go up
- Muscles to become tense
- Arteries to go into spasms

All these occur due to release of adrenaline to cope with the situation. Many patients have reported sudden blood sugar spurts after brief episodes of severe stress. The normal glucose homeostasis in the body is achieved by a delicate interplay of various hormones. The body releases adrenaline, non-adrenaline and cortisol

hormone that raise blood glucose levels to provide a quick source of energy for coping with stress.

In acute cases of stress blood glucose levels may rise quite profoundly and in extreme cases diabetic ketosis and coma also may result particularly in those with a genetic predisposition.

In chronic stress the blood sugar level is elevated due to the increased secretion of the catabolic hormones.

7.4 Infections

Infections cause a non-specific out pouring of catabolic hormone, which antagonizes insulin action, and they may trigger the onset of the disorder.

There is increasing evidence that type1 diabetes especially in the younger patients follow a Coxsackie or other virus infection. There is sometimes a long interval between the infection and onset of symptom. The virus may trigger an autoimmune reaction in the pancreatic islets and this impairs insulin secretion and ultimately destroys the β cells. Viral hepatitis can also destroy the β cells.

7.5. Diet

7.6. Age

The prevalence of diabetes increases markedly with age. Maximum incidence of NIDDM type of diabetes occurs above the age of 35 years. High proportions of Indians develop NIDDM at much younger age and therefore the prevalence of maturity onset diabetes of young (MODY) is higher in India.

7.7. Physical activity

Prevalence of diabetes mellitus is generally higher in individuals with sedentary habits. Several perspective and retrospective studies have shown the beneficial effects of physical activity on insulin sensitivity and glucose intolerance.

7.8. Nutritional status

Strong correlation was observed between rates of diabetes and mean percent body weight in a larger scale cross sectional co-countries study. Since 20-30%

diabetics in developing countries are undernourished, several claims have been made that undernourished are as susceptible to diabetes as overweight. It is suggested that weight loss and under nutrition may be a consequence of diabetes rather than the cause. It has been seen that under nutrition even during foetal life and infancy increases the risk for diabetes. There is a need for more prospective studies to evaluate the rate of under nutrition in the aetiology of diabetes.

8. METABOLISM IN DIABETES

When insulin is not being produced or ineffective, the formation of glycogen is decreased and the utilization of glucose in the peripheral tissues is reduced. As a consequence glucose that enters the circulation from various source is removed more slowly and hyperglycemia follows. When blood glucose levels exceed the renal threshold, glycosuria occurs. The loss of glucose in the urine represents wastage of energy and entails an increased elimination of water and sodium. Ordinarily thirst and increased ingestion of liquids compensate for the loss of water, but interference with the intake such as occurs in nausea or through vomiting could lead to rapid dehydration. With deficiency of insulin, lipogenesis decreases and lipolysis is greatly increased leading to excess ketone formation and accumulation. The appearance of one of these ketones, acetone in the urine indicates the development of ketoacidosis. Tissue protein is also broken down in an effort to secure energy, causing weight loss and nitrogen excretion in the urine.

9.COMPLICATIONS

Diabetes can lead to several acute and long-term complications (Raghuram *et al* ,1993)

9.1.Acute complications

Diabetes is likely to develop acute complications as a result of sudden a severe decrease or increase in blood sugar levels. Under this come mainly 3 complications

- a. Hypoglycaemia
- b. Ketoacidosis
- c. Infections

a. Hypoglycaemia

It is defined as a condition in which there is a blood glucose concentration of less than 45 mg/dl

Causes

- Unpunctual or inadequate CHO in diet
- Unexpected or unusual exercise
- Insertion of alcohol along with antidiabetic drugs

Symptoms

- Sweating
- Trembling
- Hunger
- Palpitation
- Confusion
- Drowsiness
- Incoordination
- Nausea

The diabetic who develops hypoglycaemia should immediately be given sugar or glucose and a doctor should be consulted

b. Ketoacidosis

When the body cannot utilize carbohydrate to provide energy, it burns increased amounts of fat and certain amino acids. This results in increased formation of metabolic products known as ketones. When ketones produced are more than what the body can handle they accumulate in the blood resulting in ketoacidosis. This is a serious condition as the patient may go in to coma

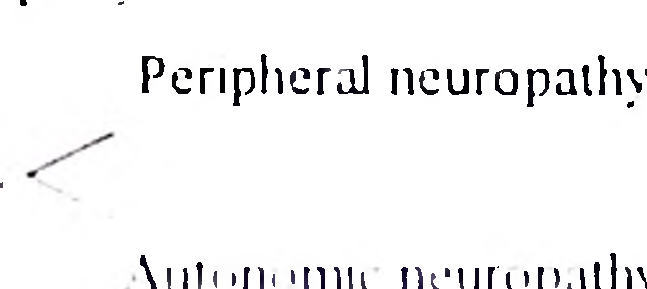
Any form of stress, particularly an acute infection or neglect of treatment due to carelessness can lead to ketoacidosis. There is intense thirst and polyuria, constipation, muscle cramps and altered vision are common. Hyperventilation with low blood pressure and acetone may be smelt in the breath. Glycosuria and ketonuria would be present, which can be detected using commercially available strips. Blood

glucose levels may be as high as 360-720 mg/dl and low plasma bicarbonate and blood pH.

c. Infections

In diabetics cuts and wounds heal slowly. They are prone to infections of skin, urinary tract and foot. In all such manifestations, the patients should promptly consult a doctor who will control diabetes and treat infections with appropriate antibiotics. Diabetes should take special precaution to prevent infection of skin and foot

9.2. Long-term complications

- a. Arteriosclerosis and heart disease
- b. Diabetic microangiopathy
- c. Diabetic neuropathy 
 - Peripheral neuropathy
 - Autonomic neuropathy

a. Arteriosclerosis and heart disease

There is an increased predisposition of the diabetic for arteriosclerosis (hardening of vessels due to deposition of fatty substances), because they generally have high levels of blood lipids, such as cholesterol and triglycerides, which make them susceptible to heart disease and stroke

The susceptibility of the diabetic to arteriosclerosis is due to several factors. Hyperlipidemia occurs in 1/3rd to 1/2 patients. HDL levels are reduced in type II diabetes possibly enhancing susceptibility to atherogenesis

Glycosylation of LDL renders it more recognizable by the LDL receptors while glycosylated HDL is more readily degradable than normal HDL, both these effects enhance atherogenesis. Diabetics have increased platelet adhesiveness and response to aggregating agents. These changes are also likely to favour atherogenesis

Most patients with type II Diabetes also tend to be obese and hypertensive. All diabetics who have had the disease for at least 10 years irrespective of the age of onset are likely to have clinically significant arteriosclerosis. Increased cholesterol levels have been recognized as a risk factor for coronary heart disease. It is

recommended that serum cholesterol should be measured in all adults' diabetics once in 2 years and in all normal of over 20yrs of age at least once in 5 yrs

Blood lipids (mg/100ml plasma)

	Desirable	Borderline-high	High risk
Total cholesterol	200	200-240	240
LDL cholesterol	130	130-160	160
HDL cholesterol	50	-	35
Triglycerides	150	150-500	500

(Ghafoomisa and Krishnaswamy, 1995)

The cholesterol level in the blood can be reduced by changing the dietary pattern or by taking drugs. The total cholesterol, HDL ratio should be < 5.

b. Diabetic microangiopathy

Diabetic Microangiopathy is characterised by thickening of the basal lamina of capillary endothelium and increased permeability of the micro vessels. This status is further aggravated by reduced deformability of the red blood cells and increased platelet adhesiveness. Diabetic Angiopathy is often considered to be a form of premature atherosclerotic disease. It usually involves peripheral vessels with thickening of the media associated with an increased risk of platelet aggregation. In many cases, the peripheral arteries of a diabetic subject feature reduced compliance.

The mechanisms by which diabetic microangiopathy develop are not known, but probably include genetic influences (Dahl, 1998). Several biochemical changes may interact, one important change being increased protein glycation. Important functional changes are increased organ blood flow, increased vascular permeability, abnormal blood viscosity and abnormal platelet and endothelial function. The structural hallmark of diabetic microangiopathy is the thickening of the capillary basement membrane. These changes may lead to occlusive angiopathy and to tissue hypoxia and damage. Screening for microangiopathy should start in children and adolescents after 5-y duration of the disease and 10 y of age. The screening should



include retinal examination through a dilated pupil or fundus photography, urinary albumin excretion rate, blood pressure measurement and neurological examination. Several intervention trials have shown that near normoglycaemia may reduce the risk of microangiopathy. There is a curvilinear association between the risk of development and progression of microangiopathy and mean blood glucose. Therefore, optimal insulin treatment is important in children and adolescents

c. Diabetic neuropathy or nerve damage

Neuropathy represents the most common complication of diabetes. Neuropathy appears to be caused by damage to small blood vessels. Nerve depends on multiple tiny vessels that carry nutrients and oxygen to keep each and every segment of these very long nerves intact. Damage to one small segment can result in loss of feeling, pain or burning sensations that bother the feet and legs.

Diabetic neuropathy almost always starts in the feet because these are the longest nerves in the body. The peripheral neuropathy that is characteristic of diabetes are found only in the presence of high blood sugar levels. High blood sugars damage both large and small blood vessels that carry O₂ and nutrients to the nerves. If there is not enough blood being sent to the nerve, the nerve's ability to work will be affected. That is why it is so important that the blood supply to the feet and legs in a diabetic not to be hampered. About 60-70% of people with diabetes already have at least a mild form of nerve damage. Diabetic neuropathy can be further classified in to two

1. Peripheral neuropathy:

- a. *Cranial neuropathy* affect cranial nerves. This leads to paralysis of nerves that control muscles of the face, for example, loss of control of the eye movements, double vision
- b. *Entrapment neuropathy* affects the hands, arms, feet or legs due to constricting ligaments
- c. *Sensory neuropathy*: decreased or total lack of sensation in various places- susceptible for burns and infections
- d. *Motor neuropathy*: loss of muscle power resulting in pain and weakness.

2. Autonomic neuropathy

- a. Urinary tract:* nerves which control the emptying of the bladder fail to function properly and the residual urine may lead to infections of the kidneys and urinary tract
- b. Sex organs-*sexual functioning for men and women may be affected
- c. Digestive system:* absorption of food or diarrhoea can occur
- d. Cardiovascular system:* heart rate and blood pressure changes may occur.

Certain organs affected by diabetes

1. Eye

Damage to the eye is the most feared complication of diabetes; Lesions affect the small blood vessels of the eyes. Lesions of the eye (retinopathy) result in rapid deterioration of eyesight. Total blindness in diabetes is uncommon since just under 2% of people with IDDM actually suffer total vision loss. Diabetes is the most common cause of blindness in the US among younger individuals, although most of this loss can be avoided with better control and through early detection of eye damage. Unless an eye exam is done, most people are unaware that they have eye damage.

2. Foot

Foot problems in diabetes can be caused by damage to both large and small blood vessels. Foot problems including nerve damage or peripheral neuropathy usually begins with vascular disease. Damage to small blood vessels in particular, appears to be the major cause of nerve damage that results in loss of feeling, or worse pain and burning sensations that bother the feet and legs. Once nerve damage progresses, it triggers loss of motor control and the abnormal gait that results in ulcers and amputations. Foot injury could go unnoticed until severe infection develops.

Preventing foot problems in diabetes begins by preventing the loss of circulation that will result in serious nerve damage. This is relatively easy today if the risks for circulating problems are recognized early. Keep BP below 130/80 is essential for reducing damage to blood vessel walls preventing plaque formation is also critical. This is done with medications. The blood flow may be improved with

high dose of vitamin E, although 1200-1500 mg a day usually required for this effect.

Signs of blood vessel problems in the feet

1. absence of foot pulses
2. pale color of the foot when it is raised
3. feet that feel cold
4. pain at rest
5. pain at night relieved by hanging the feet over the side of the bed
6. thin appearing skin
7. shiny skin
8. loss of hair from toes and feet
9. Blue color of the feet
10. reddish color of feet
11. foot infection that is hard to heal
12. ulcers that don't heal

Although amputations are 15 times as common with diabetes, about half can be prevented with simple steps that protect the feet. Preventing trauma is the best way to prevent amputation. Properly fitted shoes are essential, tennis shoes being ideal whenever possible.

3. Skin

Diabetic dermopathy This refers to roundish, slightly indented patches of skin that are brown or purplish in color. Most frequently seen on the shins, they usually occur in skin that has been injured or traumatized. Although it can occasionally be seen in people without diabetes it almost always occurs in those who are older or who have had diabetes for at least a couple of decades. Diabetic dermopathy appears to be slow healing.

Fungal infections Both fungi and bacteria feed on high glucose levels, and the skin is a favourite site for fungi to flourish. Tinea pedis (athlete's foot), which occurs between toes and occasionally the finger, is most often caused by trichophyton rubrum and trichophyton mentagrophytes. Fungal infection of the nails is

referred to as onychomycosis. Fungi do not like vinegar and vinegar soaks of the toenails can help get rid of the infection.

4. Kidney

Diabetic nephropathy is an important micro vascular complication of long standing NIDDM as well as IDDM associated with considerable morbidity and mortality. Prevalence of diabetes is on the rise and it is estimated that there are 30 million diabetics in India of which 6.6 million are expected to develop diabetic nephropathy (Ramachandran *et al.*, 1993). Further type II diabetes is 5 times more common and microalbuminuria is higher in Indians than Europeans, which cannot be diagnosed by the routine dipstick method (Gupta *et al.*, 1991). Neglect may lead to kidney failure

10. TREATMENT

Patients with 'impaired glucose tolerance' are managed at the discretion of the physician. In general, no treatment is given to elderly people, but diet and weight reduction are advised in younger subjects.

Blood glucose levels in a well managed diabetic should be less than 200mg/dl it is however, necessary to avoid the risk of hypoglycemia because it develops suddenly and threatens life. Self monitoring of blood glucose is not more effective, but is much expensive, than regular urine testing in closely supervised control programmes (Allen *et al.*, 1990)

Diabetes is managed by the following:

- a. Diet
- b. Exercise
- c. Insulin and oral drugs
- d. Education

Exercise

Exercise is a very useful measure in the management of diabetes. Exercise improves the general well being not only of normal but also of diabetic subjects. Exercise, when combined with reduced food intake, helps in reduction of body

weight and other risk factors for heart disease. It improves peripheral circulation. In addition, exercise enhances the action of insulin and thus helps to reduce the dose of antidiabetic drugs.

Persons with diabetes should therefore exercise regularly. This can include brisk walking, jogging, bicycling, swimming and playing badminton and tennis. It should become a part of daily routine. In addition, exercise is known to reduce stress and strain and enhance the quality of life. The pace and duration of exercise, however can be decided depending on the age and physical fitness of the subject. Diabetic patients on insulin before engaging in strenuous exercise should seek medical advice to prevent hypoglycaemia. They may require extra carbohydrate before, during and after the exercise

Oral drugs

In NIDDM, the body produces insulin, but this may be less effective in controlling the high blood sugar levels. Anti-diabetic tablets, in such patients either enhance the production of insulin by the pancreas or improve the action of the insulin produced in the body. There are 2 groups of drugs, which are generally used in NIDDM, sulphonyureas and biguanides. Some common sulphonyureas are tolbutamide (Rastinon), chlorpropamide (Copamide, Diabinese), glibenclamide (Euglucon, Daonil, Betanese) and glipizide (Glynase). Important biguanides are metformin (Glycophage) and phenformin (DBI). As the mode of action of these groups of drugs is different, the doctor will decide the type of drug, which is suitable to the patients.

Insulin

The doctor will decide the type of insulin that the diabetic requires. A person on insulin has to be more careful with the timings of his meals. The quantity of carbohydrate also should match the dose of insulin given. In addition to a regular schedule of breakfast, two meals and evening tea, patients with IDDM may require bedtime snacks to prevent hypoglycemia during the night. Patients with NIDDM also require insulin under certain circumstances, such as inadequate response to oral

hypoglycemic drugs and during ketoacidosis, acute infections, surgery and pregnancy.

Insulin pumps

These are pager-sized devices that infuse, programmed amounts of insulin under the skin through a catheter. These automatically infuse subcutaneous insulin continuously to maintain blood sugar control throughout the day and night. Most who go on the pump quickly come to prefer over injections but doctors warn that the device is not appropriate for everyone. It can be difficult to master, especially for patients new to diabetes who are not used to monitoring blood glucose levels and adjusting their insulin dosage before meals. In other patients, the risk of hyperglycemia may outweigh the benefits. Since the pump is continuously infusing insulin on its own, the likelihood increases that too much can be given without the patient noticing.

Pancreatic transplant (Robertson, 1992)

Pancreatic transplant and islet cell implants are still in the experimental stage. These involve enormous cost and the dangers of life long dependence on immunosuppressive drugs which have serious side-effects. Currently, most pancreatic transplantations are performed in patients with uremia and diabetes during or after kidney transplantations. A few successful pancreatic transplants have controlled diabetics.

Education

Education of the patient is an integral and important component in the management of diabetes. Nutrition counseling is the total process of individualized guidance so that the client acquires the ability to manage his/her own nutrition care, which in turn effect successful behavior change that results in more helpful behavior (Safian, 2003)

Diabetes education is a crucial part of the treatment plan. Diabetes education basically involves learning how to live with your diabetes. The diabetics should be

educated on the nature of the disease(s) they have and the possibility of development of acute and long-term complications of the disease, if blood sugar is not kept under control. Adequate basic information on diabetes enables the diabetic to comprehend and improve their psychological acceptance of the disease. In addition, the importance of following doctor's instructions regarding diet, exercise and drugs should be explained.

Diabetic should be aware of the importance of monitoring urine and blood sugar and serum lipids at regular intervals to ensure overall well-being. Patients who require insulin injections should know how to measure the insulin dose and give their own injections.

DIET

Diabetic diet need not be a complete deviation from a normal diet. The nutritional requirements of a diabetic are the same as in the non-diabetic (Raghuram, 2003). Normal Indian diet is ideal for a diabetic. However the nutrient intake has to be tailor made to the individual based on the age, sex, weight, height, physical activity and physiological needs of the patient. It is always better to consult a dietitian to prescribe and formulate a suitable individual diet. Both doctor and dietitian have a role in the dietary management of diabetes.

Hyperglycemia is the hallmark of diabetes and maintenance of normal glycemia either prevents or delays the long-term complications of diabetes. Therefore management of diabetes with diet, exercise, education and drugs has been emphasized.

The objectives in dietary management of diabetes are to

1. Reduce the blood glucose and urinary sugar
2. Maintain ideal body weight
3. Provide relief from symptoms
4. Maintain serum lipids in normal range
5. Provide all required nutrients
6. Avoid acute complications
7. Prevent long term micro and macro vascular complications

8. Improve the overall quality of life

11. DIABETIC DIET PRESCRIPTION

The nutrition and diet prescriptions is based on

- History of both the patient and his family
- Sex, age, weight, height and activity of the patient
- The type of diabetes the patient is suffering from
- Type of insulin taken by the patient, amount and when administered

Based on nature of physical activity and body weight total calorie requirement is calculated

Calories

The calculated calorie requirement should allow the patient to lose or gain weight as required and maintain body weight 10% lower than ideal/ desirable body weight

How to calculate the ideal body weight (adults)

Ideal body weight (in Kg) can be simply calculated for an individual by subtracting 100 from his/her height (in cms)

The ideal body weight shows whether the person is overweight (20% above his ideal weight) or underweight (20 % below his ideal body weight)

Based on these, the daily calorie requirements of an individual per kg body weight can be worked out

The recommended calories intake for diabetic based on body weight

Over weight - 20 kcal / kg wt / day

Ideal weight - 30 kcal / kg wt / day

Under weight - 40 kcal / kg wt / day

This is intended for person who does sedentary work. For persons engaged in greater physical activity, the recommended calorie intake may be increased as per the needs.

A person above the age of 50 years of age require 10% less calorie for each decade. If the disease is mild, a diet restricted in calories and devoid of refined sugar such as sweets, jams, and jellies is adequate.

In an obese diabetic, the calorie should be restricted to reduce body weight to ideal; after this enough calories are given to maintain normal weight. In a diabetic normal weight, enough calories should be given to maintain weight. In an underweight diabetic, enough calories should be given to increase the weight to normal and maintain it.

Maintain body weight appropriate for your height

- 1) Body weight directly proportional to calorie intake
- 2) Energy intake minus Energy expenditure = Gain in body weight
- 3) Excess CHO, protein and fat in diet increases body weight
- 4) Fat around the abdomen more dangerous than around the hips
- 5) Refined foods and empty calories from sugar increases blood sugar and triglycerides
- 6) Weight reducing diets must be low in total calories and yet ensure optimum intake of all nutrients
- 7) Weight reduction should be gradual not more than 0.5 - 1kg per week
- 8) Diet restriction should be combined with exercise

Remember, 100 Kcal in excess /day can increase the body weight approximately by 4kg in one year

Children

Nutrient requirement of children relative to their body weight are generally higher than those of adults to take care of their growth and development needs. Children suffering from Type I diabetes require the normal diet recommended for their age group. It can be calculated based on their age group. Calorie requirement for children are 1000 basal calories plus 125 cal for boys and 100 for girls for every year of age up to 12 years

Pregnant women

Pregnant women have to eat not only to meet her physiological needs but also to meet the requirements of her growing foetus. During pregnancy, the diabetic should be given 30-35 Kcal/Kg of the desirable body weight. The diet should provide 1.5-2.5 g of protein/Kg body weight. The total gain in body weight during pregnancy should not exceed 12kg. Blood sugar should be kept within normal limits throughout pregnancy to prevent diabetes related complications in the mother and newborn.

Distribution of nutrients in total calories

The total daily intake of calories from carbohydrate, protein and fat in the diet of a diabetic should be distributed in the food

Distribution of calories

	% of total calories
Carbohydrate	60-65
Protein	15-20
Fat	15-25

Carbohydrate (60-65% of total calories)

Our diet consists mainly of carbohydrate. Compared to fats and protein, carbohydrate has the greatest impact on blood sugar. One gram Carbohydrate provides 4 Kcal of energy. Generally in Indian diets, carbohydrate provides 60-70% of the total calories. Diabetics need not restrict the carbohydrate intake but they can alter the type of carbohydrate in their diet. Complex carbohydrate present in cereals and pulses are better than simple carbohydrate present in jams, jellies, sugar, jaggery, sweets etc. Complex carbohydrate should account for approximately 2/3 rd of total carbohydrate in the diet. Sugars present in fruits and milk raise the blood sugar at a slightly slower rate. Though some claims have been made that excess carbohydrate leads to hyperlipidemia and increase the risk of coronary heart disease several studies have shown that generous supply of carbohydrate (60-65% cal) in diabetic diet, when given along with high fibre diet has no adverse effect.

Rationale of high carbohydrate

Carbohydrate restriction impairs insulin sensitivity and reversed by high carbohydrate diet. High carbohydrate and high fibre diet improve insulin binding and increase in monocyte insulin receptor binding. High carbohydrate diet is likely to elevate serum triglyceride levels. Hence carbohydrate is maintained to about 50% of total calories. Most carbohydrate should be in the form of polysaccharides such as bread, cereals, and beans etc. rapidly absorbed mono and disaccharides such as sweet, chocolates and sweetened drink should be avoided.

Raising the carbohydrate intake does not adversely affect the fasting blood glucose levels, glucose tolerance or insulin requirements provided that total calories are not increased. Insulin needs are more closely correlated with total calorie intake than with the carbohydrate level in the diet.

People with diabetes should prefer carbohydrate that have low glycemic index and are high in fibre.

Distribution of carbohydrate in the diet

Since the blood sugar level depends mainly on the intake of carbohydrate it is important to distribute the intake of carbohydrate in accordance with daily needs. The total amount of carbohydrate can conveniently be divided into 4-5 equal parts.

1/3rd (33%) of diet during Lunch

1/3rd (33%) during Dinner

1/3rd (25%) during Breakfast

9% during Evening tea or bedtime

In IDDM it may be necessary to give additional CHO before the patient goes to sleep.

Glycaemic index

The rise of blood sugar after a meal does not merely depend upon the amount of carbohydrate ingested but also the rapidity of absorption, which varies with the fibre content, phytate, tannins etc. Different carbohydrate raises the blood sugar to variable extent.

The ability of the food item to raise the blood sugar is measured in terms of glycaemic index. Glycemic index indicate the extend of rise in blood sugar in response to a food in comparison with the response to an equivalent amount of glucose or any other reference food.

$$\text{GI} = \frac{\text{Blood glucose area of test food}}{\text{Blood glucose area of reference food}} \times 100$$

Foods with low glycemic index are useful for diabetics. Diets with lower glycemic index are generally rich in fibre.

Several factors such as nature of cooking, physical form of preparation, fat and fibre content influence glycemic index of different foods. Glycemic response higher with cooked than raw food. Though certain foods containing high fat such as ice cream, milk and groundnuts have low glycemic index they are not good for diabetes.

Generally, cereals like wheat and rice and root vegetables such as potato have a high glycaemic index (65-75%). Fruits have an intermediate GI (45-55%). Legumes and Lentils such as dried beans, peas etc have low GI (30-40%) and are beneficial to diabetes in moderate amounts. Bengal gram helps additionally in the prevention of arteriosclerosis by reducing serum cholesterol and triglycerides by reducing serum cholesterol and triglyceride levels. Vegetables have very low GI (20-30%).

Mean GI of some common foods

Item	GI	Item	GI
White bread	100	Orange	66
Glucose	138	Grape	62
Honey	126	Apple	53
Corn flakes	119	Ice cream	52
Whole wheat	99	Yogurt	52
Raisins	93	Whole milk	49
Sucrose	86	Skim milk	46

White rice	83	Fructose	30
Potato	81	Soya bean dried	22
Banana	79		
Sweet potato	70		

Recent studies have shown that consumption of foods with low glycemic index results in better control of blood glucose level, improvement in insulin sensitivity and HDL cholesterol levels.

Proteins (15-20% of total calories)

Protein good for health of diabetes because

- 1) It Supplies essential amino acid needed for tissue repair
- 2) Doesn't raise blood sugar during absorption as much as CHO
- 3) Doesn't supply as much calorie as fat

One gm protein provides 4Kcal of energy. Proteins taken in the diet are broken down in to amino acids before absorption in to the blood. The RDA for protein is 1gm/Kg body weight. Children, pregnant and lactating require more than this ratio of protein for growth and development and other physiological needs. It is generally recommended that 15-20% of total calorie can be derived from proteins. Protein from vegetable sources are better than from flesh foods as they add fibre and do not contribute cholesterol. In diabetes with associated renal problems protein restriction to 0.5gm/Kg body weight. However this should be of high biological values such as in egg and milk.

In IDDM children, 1-1.5gm/Kg body weight is recommended. In patients with NIDDM consumption of protein along with carbohydrate will lower the blood glucose concentration due to amino acid stimulation of insulin secretion. This helps to compensate for the defect in glucose mediated insulin secretion seen in so many of these patients. Protein also promotes satiety and helps both types of diabetic patients to adhere to the carbohydrate allowance.

72

Fats (15-25% of total calories)

Fats are concentrated sources of energy. They cannot be oxidized as readily as carbohydrates. One gram fat yield 9Kcal. Excess intake of fat thus increases body fat and leads to obesity. Fats contain essential fatty acids and are also vehicles for fat-soluble vitamins such as vitamin A, D, E and K.

The normal end products of oxidation of fats are CO_2 and H_2O . Ketone bodies are intermediated products of normal fat metabolism. They are produced in the liver and utilized by tissues to provide energy. The accumulation of these products results in diabetic coma.

In a regulated diabetic, carbohydrate cannot be utilized because of the deficiency of insulin and so the energy requirements have to be met with fats. The excessive break down of fats results in accumulation of ketone bodies, which are then excreted in the urine. Daily metabolism of about 100gm carbohydrate prevents accumulation of ketone bodies. The best way to maintain ideal body weight is to cut down fat intake.

Vitamins and minerals

These are protective factors, which in small amounts are essential for the body. Experimental studies have suggested that deficiency of vitamin B₆ result in glucose intolerance. Carbohydrates are not completely metabolized when there is a deficiency of vitamin B. It is postulated that products of partial carbohydrate metabolism like pyruvic acid accumulates in such situations and damage the nerves resulting in peripheral neuropathy. The diabetics require supplements of vitamin B. It is also advisable to supply vitamin A, as the liver, which is the storehouse of this vitamin, may be damaged in diabetes.

Deficiency of zinc, chromium, magnesium, copper results in glucose intolerance but no strong evidence to implicate these micronutrient deficiencies in carbohydrate intolerance. High fibre diets, prescribed for diabetes may interfere with the absorption of minerals such as zinc, magnesium. Similarly low calorie diets, prescribed to obese diabetics may not provide enough micronutrients and all such individuals should be supplemented with required micronutrients.

The daily intake of foods like green leafy vegetables, fresh fruits, milk and dairy products, cereals nuts, fish and egg can provide enough vitamins and minerals. Diabetes during infections and other complications may require high amount of vitamins and minerals in the form of supplements.

Fibre

It is an important component of much complex carbohydrate. It is almost always found only in plants particularly vegetables, fruits, whole grains, nuts and legumes (beans and peas). Fibre cannot be digested but passes through the intestines drawing water with it and eliminated as part of faeces content.

Dietary fibres are often recommended for the management of certain types of diabetes since they when ingested, found to reduce postprandial glucose levels in blood. In many African countries the fibre content of the diet is high and prevalence is low. In prosperous communities this relationship tends to be reversed.

Fiber rich foods slow stomach emptying and delay intestinal transit and so reduce the rate of glucose absorption, lower blood sugar rise and decrease urinary glucose excretion (Meyer *et al.*, 1988).

Fibre also contributes to satiety and the consequent decreased food intake helps reduce weight. Inclusion of high fibre food in the diet has improved control of blood glucose and lipids. Diabetics should therefore eat foods containing more fibre (35-40gm).

Fenugreek seeds

Fenugreek seed, a condiment used in Indian homes, is a rich source of fibre. It contains mucilaginous fibre and total fibre to the extent of 20% and 50% respectively. In addition it contains trigoneline- an alkaloid known to reduce blood sugar levels. Fenugreek seeds reduce blood glucose, urinary sugar and serum lipids with concomitant improvement in glucose tolerance and diabetic symptoms in both IDDM and NIDDM. The hypoglycemic action of fenugreek seeds has been shown to be due to reduction in glucose absorption and also improvement in glucose utilization at the peripheral tissues. Galactomannan present in fenugreek seeds has been shown to be responsible for its hypoglycemic and hypolipidemic actions.

Allium vegetables

The well-known member of this family, which are commonly consumed are onion and garlic other being leeks, shallot and chives. Early experimental evidences and the ones available now indicate that onion and garlic possess hypoglycemic properties. The hypoglycemic principle in onion is claimed to be 2-propenyl propyl disulphide. This principle is heat stable. At a dose of 2.5mg/Kg of body weight this compound could reduce blood glucose in healthy human subjects accompanied by increase in serum insulin level. However, when different doses of onion extract (ranging from 25-100 gm) were administered to healthy volunteers, no effect on fasting blood glucose was noted whereas in glucose tolerance test, some reduction in blood glucose was noted when given along with glucose. Use of raw as well as boiled onion showed similar effects. This suggests that onion reduces rise in blood glucose induced by glucose load, by simultaneously increasing the secretion of insulin (Polaso, 1995)

Sugars

Fructose produces a lower rise of blood sugars than glucose (Oseri, 1987). Low dose fructose boosted the ability to process glucose in the liver, an effect that could help people with poorly controlled blood glucose. However excess use of fructose is associated with triglycerides and harmful cholesterol levels.

Sugar itself either sucrose or fructose adds calories, increase blood glucose levels quickly and provides no other nutrients, people with diabetes should continue to avoid products listing more than 5gm of sugar per serving and even fruit intake should be moderate.

Artificial sweeteners

Diabetics are advised to use artificial sweeteners in place of sugar. There are several artificial sweetening agents such as saccharin, aspartame etc which are also calorie free. Their limited use is normally considered safe for diabetes.

Phytic acids/ Tannins

This usually contained in cereals and pulses, may have a more dominant role in decreasing the blood sugar rise than fibre. The blood glucose response becomes less with increasing content of phytic acid in food.

12. DIETARY GUIDELINES

In general, all foods can be classified into the following 3 categories for diabetics:

1) Foods that can be used freely

- Vegetables
- Green leafy vegetables
- Spices
- High-fibre foods

2) Foods to be used in moderate amounts

- Fats
- Nuts
- Cereals/Roots/Tubers
- Pulses
- Fruits
- Milk products
- Meat products
- Eggs
- Artificial sweeteners

3) Foods to be avoided

- Sugar
- Sweets
- Honey
- Jam and jellies
- Cakes and pastries

Sweetened juices and soft drinks

In brief, the diabetics should

- Avoid sweets
- Use fats in limited amounts
- Take cereals and pulses in right amounts
- Include high fiber foods as much as possible
- Take vegetables as desired
- Take permitted fruits in limited amounts

When diabetic patients have to eat in parties or restaurants, they should select food items from the menu based on these dietary guidelines

Model diets

1200 CAL. DIABETIC DIET (Obese)

Food stuff	Amount (g)	
	Vegetarian	Non-vegetarian
Cereals	150	185
Pulses	60	15
Green leafy vegetables	200	200
Other vegetables	200	200
Fruits	100	100
Milk	300	100
Oil	10	15
Flesh foods	-	50

This diet provides

	g	% calories
Proteins	50	16
Fats	29	21
Carbohydrates	190	63

1500 CAL. DIABETIC DIET (Normal)

Food stuff	Amount (g)	
	Vegetarian	Non-vegetarian
Cereals	225	250
Pulses	60	20
Green leafy vegetables	200	200
Other vegetables	200	200
Fruits	100	100
Milk	300	150
Oil	15	20
Flesh foods	-	70

This diet provides

	g	% calories
Proteins	60	15.5
Fats	37	21.5
Carbohydrates	224	63

2000 CAL. DIABETIC DIET (under weight)

Food stuff	Amount (g)	
	Vegetarian	Non-vegetarian
Cereals	300	350
Pulses	70	25
Green leafy vegetables	200	200
Other vegetables	200	200
Fruits	200	200
Milk	400	200
Oil	20	25
Flesh foods	-	30

This diet provides

	g	% calories
Proteins	64	13
Fats	48	22
Carbohydrates	328	65

DIABETIC DIET FOR CHILDREN

Food stuff	Amount (g)	
	Vegetarian	Non-vegetarian
Cereals	375	425
Pulses	60	20
Green leafy vegetables	200	200
Other vegetables	200	200
Fruits	200	200
Milk	300	150
Oil	25	30
Flesh foods	-	50

This diet provides

	g	% calories
Calories		
Proteins	75	13.3
Fats	60	23.8
Carbohydrates	356	62.9

Food exchange system

The quality of food and total calorie intake of a diabetic should not vary markedly, particularly for a patient with IDDM. Food exchange systems useful for this purpose in which foods providing almost same amount of calories, carbohydrate, proteins, and fats are separated grouped. This list provides variety to diet keeping the total nutrient intake constant. Different groups of food exchanges have been developed such as vegetables, fruits, cereals, legumes, flesh foods, milk and fat exchange. Each exchange list provides a number of food items that can be interchanged within the group. None of the exchange group can itself supply all the nutrients needed for a well balanced diet.

The food exchange list helps the patients

- a) Restrict the food intake according to the insulin prescription
- b) To have variety in the diet
- c) Easy learning of the principles of diet

d) To maintain body weight

List of food exchanges

- a) Milk exchange
- b) Meat exchange
- c) Fat exchange
- d) Cereal exchange
- e) Fruit exchange
- f) Dhal exchange
- g) Vegetable exchange
 {

 - Group A
 - Group B

a) Milk exchanges:

Each milk exchange contains Pro-3 g, CHO-4 g, Fat-4 g, kcal-65

Cow's Milk	- 100 ml (1/2 cup)
Buffalo's milk	- 50 ml (1/4 cup)
Curds	- 100 ml (1/2 cup)
*Skimmed milk	- 200 ml (1 cup)
*Skimmed milk powder	- 18 g (5 tsp)
Whole milk powder	- 13 g (3 tsp)
* Fat negligible	

b) Meat exchange:

Each meat exchange contains Pro-7.5 g, Fat-6 g, kcal-85

Beef	- 75 g
Chicken	- 75 g
Liver	- 75 g
Pork muscle	- 75 g
Egg	- 1 medium
Meat	- 50 g
Fish	- 75-100 g

Fish and liver contain small amounts of CHO. There is wide variation between items in this exchange

c) Fat exchange:

Each fat exchange contains Fat - 10 g, kcal - 90

Oil (any variety)	- 10 g (3 tsp)
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Ghee	- 10 g (2 tsp)
Butter	- 12 g (2½ tsp)
Vanaspathi	- 10 g (2 tsp)
Margarine	- 10 g

d) Cereal exchange:

Each cereal exchange contains Pro - 1-3 g, CHO - 18-21 g, kcals-85

Rice	- 25 g (2 Tbsp)
Cooked rice	- ½ cup
Wheat rava	- 25 g (2 Tbsp)
Broken rice	- 25 g (2 Tbsp)
Sooji	- 25 g (2 Tbsp)
Oats	- 25 g (3½ Tbsp)
Vermicelli	- 25 g (2½ Tbsp)
Flakes	- 25 g (5 Tbsp)
Wheat flour	- 25 g (3½ Tbsp)
Ragi flour	- 25 g (3½ Tbsp)
Rice flour	- 25 g (3 Tbsp)
* Arrow root	- 25 g (2 Tbsp)
* Sago	
Idli	- 1 medium size
Dosai	- 1 medium size
Chapathi	- 1 medium size
Upma	- ½ cup
Noodles Spaghetti	- ½ cup
Potato	- 100 g
Yam	- 75 g
Colocasia	- 100 g
Sweet potato	- 75 g
Taproca	- 50 g
Bread	- 2 half inch slices

* Protein negligible

e) Fruit exchange:

Each fruit exchange contains CHO - 10 g, kcals - 40

Amla	- 4 - 5
Apple	- 1 small
Apricots	- 2 fresh
Banana	- ½ small
Custard apple	- 1 small
Dates	- 2
Grapes	- 20
Grape fruit	- ½ small
Guava	- 1 medium size
Jackfruit	- 3 pieces
Jambu	- 10 small
Mango	- 1 small
Melon	- 1 slice
Orange	- 1 average
Papaya	- 2" x 3" slice
Peach	- 1 medium
Pear	- 1 small
Pineapple	- 1 slice
Plums	- 2
Sapota	- 1 small
Straw berries	- 1 cup
Sweet lime	- 1 medium size
Water melon	- 1 slice (200 g)

f) Dhal exchange:

Each exchange contains CHO - 15 g, Pro - 6 g, kcals - 85

Pulses	- 25 g (½ cup cooked)
Legumes	



h) Vegetables exchange:

Group A - Low calorie vegetables

Each exchange - ½ cup cooked vegetable CHO upto 6% kcals 30

Cabbage	- Cauli flower
Celery	- Chow chow
Lettuce	- Cucumber
Mint	- Drumstick
Spinach	- Ladies finger
Sirukeerai	- French Beans
White radish	- Capsicum
Paruppukeerai	- Kovai
Amaranth	- Knolkhol
Ash gourd	- Green papaya
Bitter gourd	- Plantain flower
Brinjal	- Pumpkin
Tomatoes	- Ridge gourd, Snake gourd,
Fenugreek	

Group B

Each exchange - ½ cup cooked vegetable CHO 6-12%, Pro - 2-3 g, kcals 50-60

Agathi	- Drumstick leaves
Beetroot	- Jack fruit tender
Carrot	- Radish-Pink
Turnip	- Onion small
Onion-Big	- Coriander leaves
Mango-ginger	- Double beans
Broad beans	- Turnip leaves
Cluster beans	- Gogu

Alcohol and diabetes

1. Alcohol contributes to calories and upset dietary regulation.
2. Alcohol damages the liver, hypoglycemia occurs due to impairment of glucose generating capacity of the liver. Alcohol is absorbed from the stomach and reaches the liver through blood stream. It is metabolized in degradation of alcohol; its capacity to release glucose from its reserve stores is blocked, thus the danger of lowering blood sugar, especially in those who are on insulin and oral drugs.
3. Alcohol induced intoxication may mask the usual symptoms of low blood glucose level.
4. Combination of a alcohol and sulfonylureas may cause intense flushing of face and palpitation. Smoking increases the risk of diseases affecting heart and blood vessels.

The diabetic should avoid alcohol. Avoiding alcohol had several advantages both for the individual and the society.

Smocking

Smockers are susceptible to myocardial infarction because

- It lowers the HDL synthesis
- Nicotine is a vaso constrictor
- Increases clotting of blood in blood vessels
- Reduces oxygen supplies to the heart and increase heart rate

Salt

Salt can raise the blood pressure and people with diabetes should limit salt intake, particularly if they have hypertension, are over weight or both. Over weight people who have a high sodium intake may be at increased risk for death for heart disease. High salt diets in people who are sensitive to its effect may harm the kidney and brain even independently of high BP. Reduction in salt intake is best achieved by cutting down the use of added salt.

Coffee and tea

Mankind's most popular drug is Caffeine. It increases the blood pressure and also produce heart beat abnormalities.

Points to be considered in planning diet for a diabetic

- a) Daily energy intake estimated considering factors as age, sex, actual weight in relation to the desirable weight, activity and occupation.
- b) Dietary calories should be 60-65% from CHO, 15-20% from proteins, 15-20% fats
- c) Simple sugars should be restricted as they have high GI.
- d) Calorie is adjusted according to type of insulin.
- e) Fat with high PUFA is preferred. Avoid atherogenic diet.
- f) High protein intake helps to increase insulin production and promotes satiety.
- g) Vitamins and minerals supplemented to meet daily requirements
- h) Timely intake of in between meal snacks should be stressed to avoid hypoglycemia
- i) Food exchange list followed
- j) Complex CHO and fiber included in the diet
- k) Patient should avoid fasting and feasting
- l) Regulation of meals needed for patients taking insulin or oral hypoglycemic agents.
- m) Sodium intake not more than 6 g daily. Sodium restricted to 3 g in hypertensive

Conclusion

Indians are generally more susceptible to diabetes more than other ethnic groups and the prevalence of diabetes is increasing with improvements in socio-economic status. Since both genetic and environmental factors play an important role the development of diabetes, at least initially, high risk population have to be screened and individuals with IGT should be advised to take appropriate preventive steps such as reduction of body weight and calorie intake, particularly from saturated fats with a increase in dietary fibre and physical activity. They are

advised to increase physical activity, maintain ideal body weight and stop smoking so that development of diabetes can be delayed, if not prevented.

NATURES' PRESCRIPTION FOR A HEALTHY LIVING

1. EAT TO LIVE, DO NOT LIVE TO EAT

Maintain a desirable body weight

2. CLEAN UP THE OILY MESS

Reduce total fat and saturated fat, avoid cholesterol and use vegetable oils in moderation

3. HE WHO FOLLOWS NATURE IS NEVER OUT OF THE WAY

Eat natural foods like coarse grains, whole grain cereals, pulses, fruits and vegetables. Restrict refined foods, processed foods and sugar.

4. A LITTLE SALT IS DIVINE, BUT TOO MUCH IS HARMFUL

Cut down salt use

5. THERE IS A DEVIL IN EVERY BERRY OF THE GRAPE

Avoid alcohol

6. DON'T GET REDUCED TO ASHES

Don't smoke

7. THE WISE DEPEND ON EXERCISE FOR FITNESS

Take regular exercise

8. HOPE FOR THE BEST AND DON'T DRIVE YOURSELVES TO DEATH

Get to know the source of your stress and avoid it. Relax!

FOOD FORTIFICATION – A STRATEGY TO COMBAT MALNUTRITION

By

**Sharon C. L.
(2004-24-02)**

**Ph.D Home Science
(Food Science and Nutrition)**

SEMINAR REPORT

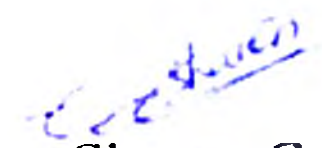
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**DEPARTMENT OF HOME SCIENCE
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2005**

DECLARATION

I, Sharon, C. L. (2004-24-02) hereby declare that the seminar entitled “**FOOD FORTIFICATION – A STRATEGY TO COMBAT MALNUTRITION**” has been prepared by me, after going through the various references cited here and has not been copied from my fellow students.

Vellanikkara

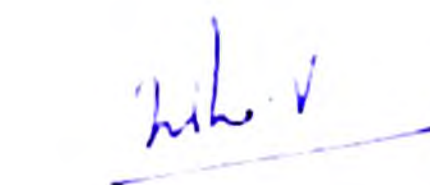

Sharon C. L.
(2004-24-02)

CERTIFICATE

This is to certify that the seminar report titled “**FOOD FORTIFICATION – A STRATEGY TO COMBAT MALNUTRITION**” has been solely prepared by Sharon, C.L (2004 – 24 – 02), under my guidance, and has not been copied from any seniors, juniors or fellow student’s seminar reports.

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CONTENTS

Sl.No	Title	Page No.
1	Introduction	1
2	Magnitudes of the nutritional problem	1-4
3	Causes of micronutrient malnutrition	4-6
4	Strategies for management of micronutrient malnutrition	6
5	Definition	6-7
6	Fortification strategy to reduce malnutrition	7-8
7	History of food fortification	8-9
8	Why is fortification done	9-10
9	Fortification technology	11
10	Fortification of foods to improve nutritional quality	11-25
11	Steps in setting up a fortification programme	25-27
12	Safety of food fortification	27-28
13	Fortification of iodine	28-32
14	Fortification of iron	32-33
15	Fortification of vitamin A	33-34
16	Multiple nutrient fortification	34
17	Indian experiences in food fortification	35-36
18	Quality control aspects of fortification	36-38
19	Limitations	38
20	Misconceptions	39
21	Conclusion	40
22	References	i-v
23	Discussion	
24	Abstract	

1. INTRODUCTION

The problem of malnutrition continues unabated despite significant achievements made in various spheres, particularly agricultural production, food sufficiency and industrial growth. High levels of malnutrition particularly among women and children have been directly and indirectly influencing mortality rates in infants, children and women. Although the country has been able to eliminate florid nutritional deficiency syndromes like pellagra, beri-beri, scurvy etc, chronic energy deficiency among adults, under nutrition among children and micronutrient deficiency across all children and micronutrient deficiency across all sections of the population continue to be a cause of concern. The use of fortification as a means of delivering nutrients to a population is more relevant and widely advocated as a means of improving the nutrition status of the population in the developing countries where there are wide spread specific micro nutrient deficiencies like iron, iodine and vitamin A

2. MAGNITUDES OF THE NUTRITIONAL PROBLEMS

2.1 VITAMIN A DEFICIENCY (VAD)

Vitamin and its derived retinoids are required for vision growth, reproduction, epithelial cell differentiation and mucus secretion. Mild deficiency causes night blindness and conjunctival lesions more severe deficiency involves the cornea, resulting in keratomalacia, xerophthalmia and permanent blindness

Vitamin A deficiency is ranked along with PEM and iron deficiency anaemia as one of the three top priority nutritional disease (WHO, 1998). VAD is a public health problem in 118 countries hitting hardest young children and pregnant women in low-income countries.

Factors leading to VAD:

1. Poverty and low purchasing power for families limit the intake of animal products, fruits and vegetables other than the cheapest staples
2. Mothers deficient in serum Vitamin A bear children with low reserves. These offspring have little chance of early replenishment due to low vitamin A content in their mother's milk
3. Diet of post weaning children lacking in calories, protein and Vitamin A, which leads to PCM and more VAD
4. Repeated Gastrointestinal infections, respiratory infection and childhood disease aggravate malnutrition and in a certain proportion of children, bring about xerophthalmia as well

Prevalence

Children (1-5 years)- 1.5%

•Night blindness - 0.71%

•Bitot's spots - 0.70%

Pregnant women

•Night blindness - 2.76%

(ICMR, 2001)

2.2 IRON DEFICIENCY ANAEMIA (IDA)

Iron is an essential trace element, involved in 3 major functions. The delivery of oxygen for sustenance of life is accomplished by hemoglobin and myoglobin, which contain iron as an intrinsic component. As a constituent of cytochrome, iron is also needed for cellular respiration. It is involved in the detoxification of lethal peroxide species formed in the tissues. Iron is a co-factor(

the metal component) of many enzymes like cytochromes, catalase and peroxidases, which carry out several vital functions in the body (Jagadeesan and Kaladhar, 2003).

Prevalence

Children (below 3 yrs) – 74%

Expectant mothers – 85%

Adolescent girls – 90%

(ICMR, 2001)

2.3 IODINE DEFICIENCY DISORDERS (IDD)

The functional significance of iodine is accounted for by its presence in the thyroid hormones, which regulate a wide variety of physiological process in virtually all tissues of higher organisms. Essential iodine is required for growth, cell differentiation and induction of RNA and protein synthesis (Jagadeesan and Kaladhar, 2003).

Goiter and cretinism are the two outstanding clinical manifestations of endemic iodine deficiency. Since a wide variety of physical and neurological disorders are associated with IDD, Hetzel introduced the term 'IDD' in place of endemic goiter in 1987.

The spectrum of IDD

Foetus: abortions, stillbirth, congenital anomalies, increased prenatal mortality, increased infant mortality, neurological cretinism, myxoedematous cretinism, psychomotor defects

Neonate: neonate goiter, neonate chemical hypothyroidism

Children and adolescents: goiter, juvenile hypothyroidism, impaired mental function, retarded physical development

Adults: Goiter with its complications, hypothyroidism, impaired metal functions

Prevalence

IDD in children (6-12)

Goitre present: 4.78 %

Grade I goitre: 4.66 %

Grade II goitre: 0.12 %

Deaf-mutism/Cretinism: 0.18 %

Adults

Goitre: 5 %

(ICMR, 2001)

3. CAUSES OF MICRONUTRIENT MALNUTRITION

In most developing countries, the adequacy of food supplies at the national level doesn't ensure that adequate food is available at the regional, household or individual level. Factors that can influence the ability of an individual to acquire and utilize nutrients include local food and water availability, food prices, a country's capacity to impart food, income and purchasing power, women workload and education level, local customs and food taboos, sanitary conditions and health status. Thus because these social, political and economical factors contribute to malnutrition, solutions require more than the provision of food and nutrients. There are interrelationships among malnutrition, poverty and economic development. The causes can be immediate, underlying or basic

Immediate

- ❖ Low intake of foods rich in micronutrients
- ❖ Low intake of substances like Vitamin C that enhance absorption
- ❖ High intake of factors like phytates and tannins that inhibit absorption
- ❖ High incidence of measles, diarrhoea and parasitic infections
- ❖ Maternal deficiencies

Underlying causes

- ❖ Inadequate breast-feeding practices
- ❖ Inadequate or incorrect complementary feeding practices
- ❖ Inadequate caring capacity: time, knowledge etc
- ❖ Low levels of family education, awareness, knowledge and motivation
- ❖ Intra-household maldistribution of access to food, health services and cares
- ❖ Poor cooking, food preparation, storage, preservation and processing facilities at household levels
- ❖ Beliefs and practices that restrict access to certain foods for family members
- ❖ Lack of institutional capacity in nutrition or personnel trained in the various components of micronutrient deficiency preventive programmes
- ❖ Low production of micronutrient rich foods
- ❖ Lack of household level gardening
- ❖ Insufficient marketing for key foods
- ❖ Poorly developed commercial food processing industry

Basic causes

- ❖ Lack of resources to produce micronutrient rich foods
- ❖ Failure to consider in agricultural and health policy making
- ❖ Poor economic or physical access to markets

- ❖ Little or no productive land
- ❖ Lack of access to seed and other inputs
- ❖ Lack of access to water for drinking, hygiene, irrigation
- ❖ Seasonability of food availability
- ❖ Low status of and lack of resource control by women
- ❖ High prevalence of certain endemic diseases

4. STRATEGIES FOR MANAGEMENT OF MICRONUTRIENT MALNUTRITION

Strategies for the prevention of micronutrient deficiencies may vary among population groups and will depend on detailed assessments of the prevalent deficiencies and their causes. To plan, implement and evaluate programs cost effectively, it is essential to have information on food consumption pattern, as well as socio-cultural and economic factors that influence both intake and metabolic need. International organizations active in micronutrient projects (WHO, UNICEF, IACG, INAVG) recommended four key strategies to eradicate micronutrient malnutrition. These are

- a. Nutrition education
- b. Dietary diversification
- c. Dietary supplementation
- d. Food fortification

5. DEFINITION

•Food fortification has been defined as the addition of one or more essential nutrients to a food, whether or not it is normally contained in the food for the purpose of preventing or correcting a demonstrated deficiency of one or more nutrients in the population groups (FAO/WHO, 1994)

- **Fortification** refers to the process of addition of a nutrient to a food to improve its quality or as a means of delivering the nutrient to a population to correct the existing nutritional deficiency among them (Rao, 2003)

Other terms related to fortification

Restoration: Addition to a food of essential nutrients which are lost during the course of good manufacturing practice, or during normal storage and handling procedures, in amounts which will result in the presence in the food of the levels of the nutrients present in the edible portion of the food before processing, storage or handling (FAO/WHO, 1994)

Enrichment Term used interchangeably with fortification, but else where it has been defined as the restoration of vitamins and minerals lost during processing (Hoffpauer and Wright, 1994)

6. FORTIFICATION STRATEGY TO REDUCE MALNUTRITION

Effective nutrition interventions are available to present malnutrition and their consequences. Short-term strategies such as nutrient supplementation have been effective in providing immediate relief but there is concern that this approach is not sustainable in the long term. Food fortification is more cost effective and sustainable solution. It plays a major role in improving the diet and meeting the micronutrient needs of the population. This must be viewed as part of an integrated food-based strategy. Others includes dietary diversification, homestead production and improved food processing and storage.

Advantages of fortification over other interventions

- Doesn't necessitate change in dietary pattern of the population
- Deliver a significant proportion of the RDA for a number of micronutrients on a continuous basis
- Can be implemented relatively quickly and can be sustained over a long period of time
- Dovetailed in to the existing food production and distribution system
- Food fortification is a population-based approach
- Food fortification is preventive
- Food fortification is cost effective

7. HISTORY OF FOOD FORTIFICATION

The Persian physician Melanpus, who suggested adding iron fillings to wine to increase soldier's potency, mentioned nutrient supplementation of foods for the first time in the year 4000 B.C. In 1831, the French physician Boussingault urged adding iodine to salt to prevent goiter. The concept of food fortification with vitamins and minerals was documented over 50 years ago to prevent overt and sub clinical deficiencies of vitamin, minerals and trace elements. Earliest reported example is the iodization of table salt in Switzerland since 1923 to prevent goiter and cretinism, which were wide spread throughout the alpine region until then. Rickets, caused by vitamin D deficiency, was once common in young children in Northern Hemisphere because of the lack of sunshine in the winter months. It was prevented by the addition of vitamin D to infant formulas and vitamin D fortification of milk and dairy products. Margarine was the first substitute or imitation food produced on a large industrial scale. Its introduction in Denmark in the 1930s led to wide spread deficiency of vitamin A in children. It was soon recognized that in order to be of nutritional equivalence to butter, margarine had to be fortified with vitamin A. Vitamin D was also added later. Flour enriched was first introduced in USA during World War II to prevent

pellagra and sub clinical deficiencies of thiamine, riboflavin and niacin. Fortification of sugar with Vitamin A, first introduced in Guatemala in 1974, is a more recent example of a large-scale nutritional intervention to prevent nutritional blindness and sub clinical Vitamin A deficiency

In India, possibilities of fortifying salt with lysine, iron and vitamin A were tried as early as in 1970. Central silk marine research institute (CSMRI), Bhavnagar, developed a process where gypsum (CaSO_4) which is already present in sea water can be co precipitated with NaCl in a single operation. Fortification at the level of 5% CaSO_4 proved successful at the production and sensory tests. The observations suggested that the utilization of Ca from CaSO_4 added to salt were similar to that of CaCO_3 and calcium lactate (NIN, 1970).

8. WHY IS FORTIFICATION DONE?

- ❖ To restore nutrients lost during processing
- ❖ Add nutrients that may not be present naturally in food
- ❖ To standardize the contents of nutrients that show variable concentration

What foods may be fortified?

The primary requirements for any fortification strategy are, the food should be carefully processed and distributed and the nutrient should be added at the point of processing. This forms a limited commercial objective of protecting the nutritional status of the consumer and conforming to the country's food legislation. In cases of fortification to improve the quality of foods, the essential requirement is that the food concerned should be centrally processed with appropriate technology to permit the introduction of the nutrient in to the product. Such additives are often advocated for cereals, which are deficient in one or more nutrients and form a bulk of the diets of the population. It is feasible in countries where all the cereals are processed, packed and marketed, but not in developing

countries where cereals are not subjected to high technology processing but mostly consumed after home level processing.

Specific criteria should be met when choosing the appropriate food vehicle to introduce the fortificant. The international life sciences and food and agriculture organization recommended that the food vehicle meet the following criteria:

- The Food should be consumed by all population groups or by majority of the target population that is at risk or vulnerable to nutritional deficiencies
- The Food should be used regularly and in consistent amounts
- The taste, appearance and smell of food shouldn't change
- The Fortificant should remain stable under extreme conditions such as cooking, food processing, delivery and storage
- The Food should not be consumed in amounts that would present a risk of consumption at toxic levels of the fortificant
- The Food should not increase the market cost in order to ensure affordability to the general population

When is a nutrient considered an appropriate fortificant ?

- Stable in the food vehicle under normal conditions of storage, distribution and use
- Physiologically available from the food
- Present at a level in which there is a reasonable assurance that consumption of the food containing the added nutrient(s) will not result in an excessive intake of the nutrient(s), considering cumulative amounts from other sources in the diet
- Suitable for its intended purpose and in compliance with applicable provisions of R.A. 3710 and regulations governing the safety of substances in foods



9. FORTIFICATION TECHNOLOGY

Advancements in science and technology have enabled the technologist to fortify almost all foods. Foods can be fortified with nutrients either in powder form or liquid form. Micro-encapsulation techniques enable liposoluble vitamins to be produced industrially in a hydro-dispersible powder forms. The technique of food fortification depends on the food processing technology used and are as follows:

1. Dry mixing for foods like cereals flours and their products, milk powder, beverage powder etc
2. Dissolution of water; for liquid milk, drinks, fruit juices, bread, pastas, cookies etc
3. Spraying for corn flakes and other processed foods requiring cooking or extrusion steps that would destroy vitamin activity
4. Dissolution in oil for oily products like margarine
5. Adhesion for sugar fortification, vitamin A in powder form is adsorbed on to the surface of sugar crystals when used with a vegetable oil
6. Coating for rice, the vitamins sprayed over the grain must be cooked to avoid losses when grains are washed before cooking
7. Pelleting for rice, the vitamins are incorporated in to pellets reconstituted from broken kernels

10. FORTIFICATION OF FOODS TO IMPROVE NUTRITIONAL QUALITY

Some commonly consumed staples and other natural foods are deficient in one or more nutrients. Improvement in their nutritional quality by addition of the deficient nutrient can go a long way in improving the nutritional status of the population.

10.1. Rice and other whole cereal grains

Inexpensive staples such as rice, corn and wheat flour have the potential to reach large population. However, these foods are often eaten where they are grown and processed at the commercial level. This limits opportunities for fortification but also increases quality and safety challenges. Notwithstanding this the fortification of whole grain cereals remains an intriguing and challenging possibility. Examples of staple foods whole nutritional quality improved through fortification

- a) Wheat which is deficient in lysine is fortified with the amino acid lysine or with other protein rich in lysine to improve its protein quality
- b) Maize deficient in lysine as well as tryptophan and can be fortified with these two amino acids
- c) Rice, particularly the highly polished one, is deficient in B- vitamins and also its protein is deficient in threonine. Attempts have been made to enrich rice with B-vitamins and threonine

The earliest methods of rice enrichment involved the production of parboiled and converted rice. By this means nutrients from the bran layer were transferred to the starchy endosperm. The parboiling process involved the soaking of the rough rice, the application of heat followed by drying and milling. It was demonstrated that in this way 50-90% of the thiamine was retained (Misaki and Yasumatsu, 1985). The process for converted rice developed by Huzenlaub (Misaki and Yasumatsu, 1985) was similar to that for parboiling, but also employed pressure differences to facilitate transfer of nutrients. Acid parboiling, described by Kondo (Misaki and Yasumatsu, 1985) was similar to the parboiling except that it was carried out in the presence of acetic acid.

After parboiling and converting, the next methods of enrichment involved the actual addition of nutrients to the milled products. Techniques used for this have been classified into two main groups 'powder type' and 'grain type' enrichment (Hoffpauer, 1992; Hoffpauer and Wright, 1994).

In powder type enrichment, a powdered pre-blended mixture of vitamins and minerals has been added at a rate of 1, 0.5. or 0.25 oz. per 100 lbs of rice (a w/w ratio of 1:1600, 1:3200 or 1:6400). For white parboiled rice, the normal practice has been to add the premix soon after milling as the heat and moisture at the grain surface at this point facilitates adherence of the powder. A major disadvantage of this method of nutrient addition has been that 20-100% of the nutrients are lost on washing. In the USA, rice enriched in this way must bear a label stating 'to retain vitamins do not rinse before or drain after cooking'.

In the second major type of enrichment, a powdered nutrient mixture has been applied to the milled rice grains followed by coating with a water insoluble substance (Cort *et al.*, 1976; Hoffpauer, 1992). A fortified rice premix produced in this way has then been added to milled rice at a rate of 0.5%, to yield an enriched product conforming to the required standard of identity.

'Grain type' or 'coated grain' enrichment have also been carried out by spraying the premix solution onto the rice which is contained in a rotary cylinder, followed by hot air drying of the rice, application of a water insoluble sealant, addition of the iron compound and finally a second application of the water insoluble compound (Bauernfeind and DeRitter, 1991; Cort *et al.*, 1976). Water insoluble coatings, which have been reported, include an ethanol or isopropanol solution of zein, palmitic or stearic acid and abietic acid. Other coating materials have contained ethyl cellulose. They dissolve at the elevated temperatures employed during cooking (Bauernfeind and DeRitter, 1991). Using this 'coated grain' procedure, Cort *et al.* (1976) and Rubin *et al.* (1977) were able to successfully enrich rice with niacin, thiamine, pyridoxine, vitamin A, vitamin E, folic acid, iron, and zinc using expanded versions of the procedures already described. The water insoluble vitamins and minerals were added in different layers with intermittent coatings of shellac. Stability to rinsing treatments were reportedly high, with vitamin losses in the range of 0.2-1.1%.

Ferric orthophosphate (white iron) is a recommended form of iron for use in the fortification of rice (Hoffpauer, 1992). This iron compound is almost water insoluble and has been preferred for mixing with milled rice due to its white colour. When it is oxidised or contains excessive moisture it may become tan, yellow, purple and or black. Hurrel (1985) reported that the bioavailability of ferric orthophosphate varied widely from batch to batch and was highly negatively correlated with particle size as was also found with elemental iron. This fortificant compound is more expensive than anhydrous ferrous sulphate. According to Hurrel (1985) the cost of ferric orthophosphate was about six times that of anhydrous ferrous sulphate, for the same level of total iron.

In Japan, a multinutrient enriched rice has been on the market since 1981. The first step in this procedure was acid parboiling in the presence of thiamine, riboflavin, niacin, pantothenic acid and pyridoxine. The second step involved coating the grain with separate layers of vitamin E, calcium and iron in separate layers and finally a protective coating material (Misaki and Yasumatsu, 1985). Modified atmosphere packaging, utilising aluminum laminate and carbon dioxide, ensured the stability of vitamin E during storage. The yellow or brown colour of the premix was not a problem in this case as it was used in the fortification of 'brown' rice.

Apart from these well-established procedures there have been other innovations regarding alternative methods for rice fortification. Joseph *et al* (1990) described an enrichment procedure using a premix containing thiamine, riboflavin, niacin and pyridoxine. This procedure involved soaking the milled rice in an acid medium containing the water-soluble vitamins followed by the cross linking of starch granules in the enriched grains. The cross linking procedure itself was demonstrated to have caused significant vitamin loss, but the added vitamins were highly cook and wash stable. It is possible that this method could have some utility in the future.

Use of fortified simulated grains has featured prominently in attempts at rice fortification in the developing countries. Murphy *et al* (1992) described the production of such a fortified rice product for use in the Philippines. The synthetic rice grains were produced by extrusion of rice flour in a pasta machine. The best formulation contained vitamin A stabilised by a mixture of tocopherol, ascorbate and lipids with a low level of unsaturation. Retinyl palmitate stabilised in an acacia matrix, type 250 SD (Sigma Chemical Co.) was the fortificant used. Retention of vitamin A after washing was reported to be 100%. Vitamin retention after cooking, however, ranged from 60-94%. The formulations, which demonstrated the better storage stability, particularly at high humidity, suffered greater cooking losses. Drawbacks with this technology have been reported to exist with respect to blending with the natural product and in the consistency of the simulated grains after cooking (Murphy *et al*, 1992; Hoffpauer and Wright, 1994)

The enrichment of whole-wheat grains with vitamin A has been attempted (Combs *et al*, 1994). The fortificant used was a premix comprised of concentrated vitamin A attached to wheat grains, for mixing at a level of 0.25% with wheat grains. The feasibility of this procedure was not determined. The fortification of whole grain cereals with soluble iron compounds is difficult because they promote oxidation of the lipid component of the grain, thus reducing the shelf life.

10.2. Flours, cornmeal and bread and pasta

In the fortification of flour the required nutrient mixture is mixed with an appropriate diluents to produce a premix, which is then accurately metered into the flour. The addition of vitamins B₁ and B₂, niacin, iron and calcium to wheat flour is a common practice in many developed countries. It is technologically feasible to add other vitamins and minerals as well. Cort *et al* (1976) successfully used two premixes to fortify wheat flour, the first comprised vitamin A,

pyridoxine, folic acid, tocopherol acetate, thiamine, riboflavin, niacin and iron while the second contained calcium, magnesium and zinc.

The vitamin/iron premix demonstrated excellent stability on storage. The flour enriched with both premixes also demonstrated excellent stability on storage at room temperature. Under conditions of accelerated storage at elevated temperature (45 °C), however, there was substantial loss of vitamin A beyond 4 weeks of storage. Parrish *et al* (1980) also reported good stability of enriched wheat flour stored at room temperature, but about 50% losses in flour stored at 40 °C for 6 months.

The form of vitamin A most commonly used in the fortification of flour was dry stabilised vitamin A palmitate (type 250-sd) powder form. The water soluble vitamins (thiamin, riboflavin, niacin, pyridoxine, folate and calcium pantothenate) are used in pure crystalline form. The mononitrate salt of thiamine is preferred for this use. Iron is normally used in its reduced elemental form. In the work of Parrish *et al* (1980) ferrous sulphate was also used as the source of iron and there was no significant difference demonstrated between the flour enriched with ferrous sulphate and that enriched with elemental iron. It has been recommended that ferrous fumarate be used in the fortification of wheat flour within the P.L. 480 programme, due to its greater bioavailability (Combs *et al.*, 1994).

Technology in modern flour mills

- A dry powder feeder to meter out the fortificant or premix
- A means to agitate the fortificants or pre mix so that it feeds smoothly and uniformly without lumping
- A mechanical means of adjusting the rate of flow of the mix
- A conveying system to deliver the premix from the feeder to the flour

Feasibility of flour fortification

- Simple technology and well established
- Extensive experience-50+ years of history and over 30 countries currently fortify cereal flours
- Economical-very cost effective for providing nutrients

Rubin *et al* (1977) investigated the stability of bread made from flour enriched by the 6 vitamins and 4 minerals described by Cort *et al* (1976). They found that the inclusion of calcium and magnesium adversely affected vitamin A retention during the baking process. There was also an off-flavour detected in bread, which included magnesium after 5 days of storage. Crumb colour and grain were also negatively affected by the nutrient multimixes tested by Rubin *et al* (1977), with the greater affect occurring with the nutrient mix which included magnesium. Fortification of bread with zinc salts exerted no adverse effect on loaf volume, flavour or any other index of bread quality (Ranhotra, 1976)

Iodization of bread has been carried out in the Netherlands and Tasmania (Dunn *et al*, 1986) by the addition of 2-4 ppm KIO_3 to the bread improver, which was already in general use. KIO_3 has been used in bread production in the past, not as a fortificant but as an oxidising agent to improve dough quality.

The fortification of corn meal with 6:4 vitamin/iron mixture was shown to be technically feasible (Rubin *et al*, 1977, Parrish *et al*, 1980). Losses of vitamin A on storage at room temperature for up to six months were greater than with wheat flour but still remained below 20%.

In many countries pasta or noodles are commonly eaten and these can therefore be important vehicles for fortification. The manufacture of such products involves the production of dough, which is then extruded and dried. Enrichment can be through the use of enriched flour or alternatively, wet addition of a

dispersion of the required vitamins can be carried out at the dough-making stage. Vitamin losses during production depend largely on the drying conditions employed. Dexter *et al* (1982) enriched durum wheat flour with a vitamin mixture containing riboflavin, thiamine mono nitrate and niacin for the production of spaghetti. They reported that high temperature drying treatments resulted in significant losses of riboflavin, whereas the other vitamins were stable to the processing conditions. In all cases cooking losses, estimated between 40 - 50%, exceeded those experienced during processing.

10.3. Breakfast cereals

The fortification of ready to eat breakfast cereals is a wide-spread practice in developed countries. Surveys have indicated that fortified breakfast cereals play an important role in ensuring nutritional adequacy of the diets of the U.S. population

The minerals and the more heat stable vitamins like niacin and riboflavin have been added to the basic formula mix prior to processing (Johnson *et al.*, 1988). The heat labile vitamins such as vitamins A, C and thiamine are usually sprayed onto the cereals after the high temperature processes such as oven or extruder

Good storage stability of the fortified breakfast cereals was reported (Anderson *et al.*, 1976). This was partly due to the fact that packaging materials with appropriate barrier properties have to be selected to ensure the maintenance of equilibrium moisture contents below about 5 %. The addition of ascorbic acid was found to stabilise vitamin A to a greater extent than did phenolic antioxidants (Anderson *et al.*, 1976). The addition of tocopherols did not provide any additional stability, possibly because they were already present at optimal levels

10.4. Milk and milk products

10.4.1. Liquid milk

The production of a large proportion of milk on the market involves the removal of cream. Along with the cream, much of the fat-soluble vitamins are also removed. The fortification of milk commonly involves the addition of vitamins A and D.

Since milk is an oil-in-water emulsion, the possibility exists to add vitamins in their oily form or use water dispersible forms of these. Ease of mixing has been identified as an advantage of using dry, water dispersible forms of the fortificant. However, the disadvantage of this was that vitamins were less stable in this form after addition to the milk as the protective coating dissolved leaving the vitamin susceptible to degradation (O'Brien and Robertson, 1993). Addition of oily vitamin preparations was recommended after dilution and pre-homogenisation with a suitable quantity of milk. Addition of the vitamin mixture prior to homogenisation of the bulk supply facilitates uniform mixing. Metered injections of the vitamin preparation upstream to the homogeniser have been the standard set up in continuous operation plants. As both of these vitamins are sensitive to oxidation, care must be taken to minimise aeration particularly during mixing stages.

Many iron compounds have been assessed in the fortification of pasteurized whole milk. At pasteurization temperatures below 79 °C off-flavour due to lipolytic rancidity developed but these problems were greatly reduced by increasing the pasteurization temperature to 81 °C. De-aeration of the milk prior to the addition of iron compounds was also found to reduce flavour problems. The best fortification procedure was judged to be the addition of ferric ammonium citrate followed by pasteurization at 81 °C. In this way fortified milk containing 30-ppm iron was found to be acceptable after 7 days storage.

Calcium fortification of milk and milk-based beverages has been carried out. Calcium fortificant preparations including stabilisers and emulsifiers have been used for this purpose to maintain calcium in suspension so as to improve mouth feel and appearance of products. In Germany a milk-based fruit beverage has been marketed which is fortified with calcium, phosphorous as well as vitamins A, E, B and C

10.4.2. Powdered milk

The fortification of powdered milk has been achieved by the addition of dry vitamin preparations to the milk powder as well as by vitamin addition to the liquid milk just prior to spray drying. As with other dry products, effective mixing has been best achieved in two steps: the initial dilution of the vitamin mixture with a suitable quantity of milk powder, followed by mixing into the bulk. Consideration of particle size and density are important to prevent separation of the components on storage

In the iron fortification of powdered non-fat dry milk, ferrous sulphate at a level of 10 ppm was found to be stable for a period of 12 months. Ferric ammonium citrate and ferric chloride at a level of 20 ppm iron in the reconstituted product gave acceptable results (Cocodrilli and Shah, 1985)

10.4.3. Other dairy products

The addition of vitamins to other dairy products such as yoghurt and ice cream has been practiced and enrichment of cheese with iodine through the use of iodised salt has been approved in Germany.

In the case of ice cream, there were no technological difficulties to overcome. The unit operations used in the manufacture of ice cream are not highly destructive to vitamins. Vitamins are added in the dry form to the mix. Since

whipping and consequent aeration of the mix is carried out around freezing temperature, oxidative losses of vitamins are minimal. Perhaps the greatest processing losses are due to pasteurization of the mix.

In the production of yoghurt, the low pH conditions render it unsuitable as a carrier for vitamins such as vitamin A (O'Brien and Robertson, 1993). The water soluble B-vitamins are best used in a coated form, protected for odour and flavour considerations. When vitamins are added to the yoghurt by addition to the base, some vitamin loss can occur through metabolism by fermentation microorganisms (O'Brien and Robertson, 1993).

10.5. Fats and oils

10.5.1. Margarine

Margarine is a spread, which has been widely used interchangeably with butter. For this reason, in many countries fortification of this spread with vitamins A and D is practiced, since the food that it replaces is a good source of these vitamins. The vitamin A requirement is met using β -carotene as well as oil soluble vitamin A esters (Bauernfönd, 1991). The oil soluble vitamins are added in the required amounts to a portion of warmed oil, which is then added to the bulk prior to homogenisation. Particularly in the case of margarines with a high content of polyunsaturated fatty acids, vitamin E has also been added. Due to the mild processing conditions only small overages are required to compensate for processing losses: 10% for vitamins A and D and between 5-15% for vitamin E (O'Brien and Robertson, 1993).

10.5.2. Oil

Fortification of oil with vitamin A in the form of retinyl palmitate has been attempted in Brazil (Nestel, 1993). Storage studies demonstrated that after 18 months of storage in dark sealed containers losses of more than half of the vitamin content were experienced. When storage was not carried out in the dark, most of

the vitamin content was lost after 6 months. Packaging of the fortified oil in opaque containers was therefore demonstrated to be a critical consideration. Vitamin A fortified oil showed good vitamin retention after 5 months of storage in sealed metal containers at high temperature and humidity (Combs *et al.*, 1994).

10.6. Accessory Food Items

10.6.1. Salt

Salt iodisation began in 1922 in Switzerland and has been implemented in many countries as the major mechanism for eliminating iodine deficiency. Today, IDD remains a problem in many countries. WHO and UNICEF have established the goal of 'Universal Salt Iodisation' to be achieved by the end of 1995. Salt has been favoured as a carrier for iodine due to its wide spread coverage, effectiveness, simple technology involved and low cost. Based on the suitability of salt as a widely used and low cost vehicle, fortification of salt with other nutrients has also been attempted (Nestel, 1993, Rao, 1985).

According to the Codex standard for food grade salt, use can be made of potassium or sodium iodides and iodates. The iodates have been found to be more stable than the iodides under a wide range of conditions. Stability studies of iodised salt using potassium iodate as the fortificant demonstrated that there was no significant loss of iodine on storage in polyethylene bags for up to two years (Chauhan *et al.*, 1992) and that boiling of the salt solutions led to negligible iodine loss.

According to Bauernfeind (1991), an acceptable iron fortificant for salt is one, which does not discolour the salt, nor impart a flavour or odour and remains stable and bio-available on storage.

Nestel (1993) reported on the use of a fortificant mixture containing 40 ppm of potassium iodate, and 1000 ppm of iron as ferrous sulphate and 10,000

ppm of a permitted stabiliser, which rendered good bioavailability of both iodine and iron after prolonged storage. Cost was identified as a constraint in the use of this fortificant system as it added 50% to the retail price of salt.

Fortification of salt with vitamin A has been attempted under laboratory conditions (Nestel, 1993). The fortificant used was dry vitamin A palmitate type 250 SD protected by a lipid. The fortificant was found to be unstable at moisture contents above 2%, since salt is hygroscopic, packaging material with an adequate moisture barrier must be used. Impurities in the salt were also found to destabilise the vitamin A. The particle size and shape must be such that uniform mixing could be achieved and segregation does not occur on storage.

10.6.2. Sugar

Sugar has been found to be a suitable vehicle for nutrients in fortification programmes in Latin America and the Caribbean. In the vitamin A fortification of sugar, vitamin A 250-CWS was proven to be the most effective fortificant. Fortification of sugar with iron has also been attempted. Bauernfeind (1991) reported promising results using sodium ferric EDTA as the fortificant. Segregation of the fortificant and the carrier was not a problem as the iron compound became stuck to the sugar crystals at moisture contents exceeding 1% (Cook and Reuser, 1985). A major problem, which exists with iron-fortified sugar, is that on addition to coffee or tea, there is marked discolouration. This phenomenon is reduced with the use of NaFeEDTA, but it is still evident.

10.7. Beverages

10.7.1. Tea

Brooke and Cort (1972) have reported on two procedures for the fortification of tea. Fine powdered vitamin A palmitate 250 SD was used by dry

mixing with the tea dust. An emulsion of vitamin A palmitate, diluted with 50% sucrose solution was sprayed onto tealeaves. The added vitamin showed excellent storage stability for periods up to 6 months, and showed 100% recovery after brewing.

10.7.2. Fruit juices and drinks

In most cases the pH of fruit drinks and juices is below 4.5 and the heat treatment required is pasteurisation. Some loss of heat labile vitamins, thiamin, folic acid and ascorbic acid, occur as a result of the thermal treatment. The acidity of these drinks causes problems of stability with vitamin A, folic acid and calcium pantothenate. Carbonation of these beverages, with the resultant exclusion of oxygen, improves the stability of vitamins. The presence of sulphur dioxide in the fruit juices used in the production of these beverages has been shown to have a detrimental affect on thiamine content (O'Brien and Robertson, 1993).

Frozen orange juice concentrate containing added vitamins A, B₁, and C showed no vitamin A loss after holding at -10°F for 6 months (DeRitter and Bauernfeind, 1991). Beta-carotene and apocarotenal both showed good stability in fortified drinks but the latter was less stable on exposure to sunlight.

Problems associated with the iron fortification of fruit juices and drinks have been outlined as follows (Coccodrilli and Shah, 1985)

- i. Accelerated loss of vitamin C
- ii. Flavour and taste deterioration in the presence of thiamine, folic acid, vitamin A and vitamin C
- iii. Levels of fortification beyond 2.7 mg per serving result in metallic off-flavours
- iv. Decolourisation of some pigments

The stability of a powdered breakfast drink fortified with iron, vitamin A and vitamin C was found to be very good. For organoleptic considerations ferrous gluconate was found to be superior to ferrous sulphate

10.8. Infant formulas

An adapted formula is designed to supply the total energy and nutrient requirements of full-term healthy infants during the first year of life. There must be careful selection of mineral compounds added to the formulas, as cereal products are highly susceptible to lipid oxidation during storage. In a study of iron fortification of infant cereals, Hurrell *et al* (1989) proposed the use of ferrous fumarate and ferrous succinate as they gave rise to no objectionable flavours, odours or colours on storage. Ferrous sulphate coated with hydrogenated fats, mono- or di-glycerides and ethyl cellulose caused discolouration on reconstitution with hot milk and hot water.

Although some allowance is made for the natural vitamin content of the ingredients used, most of the vitamins are added to the formula. The Codex Alimentarius Commission (FAO/WHO, 1994) has published an advisory list of mineral salts and vitamin compounds which can be added to formulas.

Iron absorption from formulas has been reported to be 5-10% compared to 50% for human milk. It has been suggested that bovine milk proteins or elevated calcium and phosphorus levels account for this difference. Zinc levels in formulas are also higher than in human milk to make up for reduced bioavailability.

11. STEPS IN SETTING UP A FORTIFICATION PROGRAMME

When the decision for food fortification as a suitable strategy against micronutrient malnutrition has been adopted, the following steps in the design of a fortification programme can be distinguished

- i. Identification of the target group
- ii Identification of the nutrient to be added
- iii. Selection of foods to reach the vulnerable i.e.: the target group
- iv .Level of nutrients to be added
- v. Execution of test protocol: laboratory testing, bioavailability and pilot trails

Preliminary laboratory trials must be carried out to determine stability and compactability of the chemical from of the nutrient with the vehicle e.g. vitamin A compatible with dry foods like sugar and condiments but not with salt

Metabolic studies carried out to determine the absorption of the nutrient from the fortified food fed along with the habitual diet

Determine the compactability of added nutrients with the food and acceptability by the target population

Testing the impact of continuous consumption of the fortified food on the status of nutrient concerned among the target population under realistic field condition

Pilot and Large-scale production of these fortified foods

Necessary legislations and quality standards for the fortified food and a system of monitoring its nutrient content have to be set up before it is marketed commercially among the population

Who shall be responsible for the Food Fortification Program?

Since micronutrient is a global consumer need, a grand alliance among food industry, government, international and non-government organizations is urgently needed to eliminate micronutrient deficiencies. Each one can help eliminate micronutrient malnutrition through affordable food products to which are added very small quantities of micronutrients. Their potential contributions are as follows:

INDUSTRY (to do the fortification)

- technical know-how; use of appropriate technology; use of, if possible, local ingredients; quality control, quality assurance
- marketing & distribution skills; targeting groups and finding effective ways of reaching them
- post-marketing surveillance
- use of initial margin reduction as investment
- local nutrition education programs

GOVERNMENT (to assure fair trade and to protect consumers)

- advocacy, policy-making, regulatory functions, standards-setting and enforcement of quality assurance
- provision of tax breaks, incentives for validated health claims

NON-GOVT. ORGANIZATIONS-NGOs (to provide supportive role)

- advocacy and promotion of positive attitudes for fortification organizations at the global, national and community levels
- endorsement of standards and product categories
- promotion of products that conform with standards
- training of health, education, and other key workers

12. SAFETY OF FOOD FORTIFICATION

A preferred intervention in the management of micronutrient is through a diet that provides a safe concentration of intakes. As a matter of safety, it is advisable to give near physiological dose daily, which doesn't require strict medical supervision (Subbulakshmi and Naik, 1999). Except for vitamin A and other fat-soluble vitamins, other vitamins are non-toxic even if ingested at levels higher than the recommended intakes. Level of food fortification generally ranges b/w 15% and 25% per serving which is much below the critical levels. E.g. Vitamin C consumed in amounts up to 100 times the RDA for very long periods without any undesirable side effects.

How much nutrient should be added to foods

The amount of nutrient(s) to be added is as follows:

- For essential nutrients that are deficient in the diet, the added nutrients shall be 1/3 of the RDA of the target consumer except vitamin C, which shall be supplied at not less than 100% of the RDA in fortified juices/flavored drinks;
- For nutrients that are essential but have not been established to be deficient in the diet, the added nutrients shall supply at least 1/5 (or 20%) of the RDA of the target consumer'
- For nutrients that are essential but have no established RDA, the added nutrients shall supply at least 20% of the estimated safe and adequate levels for the daily intake,
- For processed foods to be fortified with nutrient(s) with known toxicity (e.g. vitamins A, D, E, K, Zn, Se), the level of such nutrient(s) in the food shall not exceed 150% of the RDA for the target consumer per prescribed serving likely to be consumed per day.
- For essential amino acids, fortification levels shall be in accordance with the recommendations of the Joint FAO/WHO/UNU Expert Committee on Energy and Protein Requirements, and,
- For nutrients that have not been established as essential for humans, fortification shall be at a significant level above the natural state as determined by the precision of the analytical methods

13. FORTIFICATION OF IODINE

Salt is one of the most suitable vehicles for iodine fortification and in general, safely used for over 70 years in programmes around the world to prevent iodine deficiency problem. The most commonly used compounds in the iodisation of foods are the iodides and iodates of sodium and potassium. These are the additives allowed by the Codex Alimentarius in the iodisation of salt. The iodide compounds (Bauernfeind, 1991) are cheaper, more soluble and have a higher

iodine content (so that less is needed to achieve the same level of iodisation) than the corresponding iodates. Iodates are more stable under conditions of high moisture, high ambient temperature, sunlight, aeration and the presence of impurities. The use of iodate is therefore recommended for use in developing countries. Potassium iodide is well suited in cases where the salt is dry, free from impurities and has a slightly alkaline pH. Otherwise the iodide may be oxidized to molecular iodine and lost through evaporation. If excess water is present the iodide may be separated from the salt in the water film (FAO/WHO, 1994). Loss of iodide can be reduced through the addition of stabilizers such as 0.1% sodium thiosulphate and 0.1% calcium hydroxide combined or 0.04% dextrose and 0.06% sodium bicarbonate (Kuhajek and Fiedelman, 1973). Calcium salts have been used some report of off-flavour due to the calcium ions. The calcium compound is also much less water soluble than sodium and potassium compounds and this further limits its applicability.

The level of fortification that has been used ranges from 30-200 ppm. Daily consumption of 10 gm of iodated salt (25 ppm of potassium iodate), provides about 150 µg of iodine. Using iodinated salt can effectively control goiter endemias of mild to moderate degree.

Oil fortified with iodine is available for oral or intra-muscular injections. France is the only country in the world, which is producing on a commercial scale, iodised oil from poppy seed oil for injection (Lipidol) as well as for oral administration (Oriodol). An intramuscular injection of 1 ml of iodised oil containing 480 mg of iodine can maintain satisfactory iodine supply for 2-3 years, while the oral dose of 1 ml can give protection for one year. China is also producing iodised oil for oral use to meet its domestic requirement.

Supplementation of iodised oil is the best method of immediate prevention of new cases of cretinism and mental retardation in the communities living in

severe iodine-deficient areas. Once the iodised salt distribution programme is well established, the iodised oil programme can be slowly passed out in these areas.

Universal salt iodisation (USI)

The UNICEF-WHO joint committee on health policy met in Geneva in Jan 1994. It briefly reviewed the progress in IDD, and sharpened the focus on iodised salt.

The Highlights of the committees report:

- i. The current intermediate goal is now to iodize all salts for human and animal consumption (including salt for food processing) in all countries where IDD is a public health problem
- ii. IDD assessment is necessary to know if the problem exists, but great detail is not necessary in order to act towards salt iodisation
- iii. The levels of iodine in salt should be adjusted after system is in operation and monitored by urinary iodine levels of people living in the main risk areas
- iv. The steps towards salt iodization include
 - a. Identifying major salt sources
 - b. Setting standards, regulation and legislation
 - c. Establishing advocacy and mobilization plans
 - d. Conducting feasibility studies of USI
 - e. Establishing procurement and installation plan
 - f. Assuring adequate supplies of iodine
 - g. Establishing internal and external quality control procedure
 - h. Developing and education campaign for iodised salt
 - i. Mobilizing necessary resources to achieve effective iodisation
- v. Monitoring is recognized as essential to a sustained programme

IDD prevalence and control programme data: Indian scenario

Ministry of health and family welfare in collaboration with the salt commissioner in the ministry of industry ensures the production, distribution and quality control of iodized salt. There is a Central IDD control cell at the Directorate General of Health Services (DGHS), and each state has its own IDD control cell. State health department are responsible for quality control of salt within state, creating consumers demand, monitoring iodized salt consumption, training, information, education and communication. India is self sufficient in its salt production.

It is now estimated that over 70% of salt consumed in India is iodised. The scale of salt iodisation activities underway in the major salt producing regions of the country is very impressive. In addition to the commitment of the government of India to this initiative, the existence of a separate entity-the salt commissions office to organize and co-ordinate the development of the salt industry, improving manufacturing methods and overseeing the distribution of salt throughout the country deserves mention. The co-operation and collaboration of the salt producers, who understand the importance of eliminating IDD and of their role in improving public health has resulted in rapid progress towards USI. The production of large salt crystals, which posed a problem for the iodisation programme, has been largely discontinued. Although large crystals could be iodised by spraying with iodate solution, they were difficult to pack and the bags often get torn. During storage and transport, if dust and dirt accumulated on the crystals, consumed would end up washing the salt, thereby washing away the iodine on the surface of the crystal and thus very little iodine would actually reach the consumer.

A combination of consumers demand and the salt commissioner efforts has made producers, to modify their production techniques to produce smaller crystals. There is an increasing demand and supply of salt packed in 1 kg PE bags.

Presently, there are approximately 9000 common salt producers in India, mainly in the private sector (90%), who until recently were partially subsidized by the government. There are around 500 iodination plants for commercial production of iodised salt. Salt is produced using sea brine, sub soil brine and lake brine. Public producers like Sambhar salt lake and Hindustan Salt Limited produce salt from lake and sub soil water.

14. FORTIFICATION OF IRON

Fortification of foods with iron would act as a long-term measure to improve the iron balance in the entire population. Salt has been found to be the most suitable vehicle for this purpose since its cheap and universally consumed. Initial trials have proved the efficacy of the iron- fortified salt in improving the iron status of rural population. Fortification with iron has been successfully adopted for wheat flour, rice (Flaventino and Pedro, 1990), sugar (Zoller *et al.*, 1980), milk (Walter, 1990), fish sauce (Cook and Reusser, 1983) and curry powder (Lamparelli *et al.*, 1987). Other foods like wheat biscuits, wheat flour noodles and maize meal have been tried.

It is important to assess the contribution of iron fortification in combination with other strategies for the control of iron deficiency in specific age/sex groups, so as to establish optimal goals for iron fortification of foods. Furthermore, it should be stressed that not all types of anaemias are due to iron deficiency.

Iron bioavailability, influenced by physical and chemical properties of the fortificant as well as the presence of substances, which either inhibit or improve iron absorption, there is a need to develop convenient models for the evaluation of iron bioavailability.

The major chemical characteristics of iron sources that determine their behaviour in foods are

1. Their solubility e.g. ferrous salts more soluble than ferric
2. Their oxidative state: ferrous salts are most effectively utilized than ferric salts and tend to be more reactive in food system
3. Their ability to form complexes, which are not bio available: generally ferric iron has a greater tendency to form chelates than ferric iron
4. The presence of metal ion such as iron can speed up vitamin degradation and loss of nutritional value of a food product particularly for vitamin C, thiamine and retinal.
5. Catalyze the oxidative rancidity of oils and fats
6. Produce undesirable colours, colour fading from off-flavours and undesirable precipitates.

Young children aged 6 to 18 months, pre school age children, women and adolescent girls are most vulnerable to iron deficiency anaemia. Iron EDTA is not currently approved for use in foods in US because total EDTA levels in the diet from other types of EDTA used in processed foods approach allowable limits. Iron EDTA is more bio-available form of iron compared to some other forms. Nevertheless, in developing countries where people consume much less total dietary EDTA, it should be seriously considered as an attractive iron fortificant.

15. FORTIFICATION WITH VITAMIN A

National level vitamin A fortification is indicated when at least two of the following 3 criteria are met

- 20% or more pre school children have serum retinol levels below 20mcg/dl
- 20% or more lactating women have breast milk retinol below 30mcg/dl
- 20% preschool children consume < 50% of their RDA for vitamin A.

In countries like Guatemala and Costa Rica sugar is used as a vehicle for fortification. In India foods like vanaspathi, bread, and milk are fortified with vitamin A in a limited scale. In both India and Pakistan, fortification of tea was considered. Both tea dust and tealeaves were successfully fortified in India. Moisture contents in excess of about 7-8% in a food are known to adversely affect the stability of vitamin A.

There is essentially no risk of vitamin A deficiency or toxicity, when dietary intake from food is less than 10,000 IU vitamin A / day. The safety range is 10 times the RDA (Nilson, 1994). Under normal physiological conditions, however, significantly higher levels of retinal intake just before or soon after conception may have teratogenic consequences. For this reason, WHO recommended that daily supplements of 3000RE (10,000 IU) vitamin A can be given safely any time during pregnancy.

16. MULTIPLE NUTRIENT FORTIFICATION

It is well known that nutrient deficiencies do not occur in isolation. The inter relationship of various nutrients points to the fact that a single nutrient supplement is not going to alleviate the widespread micronutrient deficiencies. Considering the complexity of the problem of malnutrition, a multi-pronged approach is needed. In this regard, it makes sense to add more than one nutrient, while opting for staple food fortification. It may marginally increase the total cost of fortification but the advantage of doing so is far more beneficial.

While flour and corn meal have been tried in developing countries for fortification with vitamins and minerals (Cort *et al.*, 1976). Organoleptic properties of products made from these flours were known to be on par with their conventional counterparts. Addition of magnesium and calcium in these flours has to be technically improved upon due to their ability to impart off-flavour and colour.

17. INDIAN EXPERIENCES IN FOOD FORTIFICATION

Interesting aspect of food fortification in India in earlier days was that the fortified foods were for prevention rather than cure. Thus, foods tried were wholesome foods e.g. Indian multi purpose food, synthetic rice, bread etc. lysine supplementation of cereal diets dominated initial phases of fortification in India (NIN, 1970). Later on, the focus has been shifted to single nutrient fortification as a curative measure for specific nutrient deficiency. Now with the realization that single nutrient deficiency is inter-linked with other nutrient deficiencies, reversal of earlier concept of multi-nutrient fortification has surfaced.

Synthetic rice

- Fortification of starchy material with high protein source tried by scientist in CFTRI
- Project was successful both nutritionally and technologically, but establishing plant didn't succeed

Wheat flour

- Wheat flour with edible peanut flour -CFTRI
- Acceptability problems in chapatti

Bread

Bread with lysine and vitamins and a few minerals
 Significant improvements in the weight and heights in children
 Now soy flour is used

Salt

Project for salt fortification was undertaken by the food and nutrition board of the government of India in collaboration with the National Institute of Nutrition.

- NIN recommended the fortification of salt with iron (ferric orthophosphate and sodium acid sulphate, at concentration that supplies 1mg iron per g of salt)

•Was acceptable and improved the haemoglobin status and reduced prevalence of anaemia

Other foods

Sugar fortified with iron was studied in detail by Disler *et al* (1975) and Layrisse *et al* (1976) and proved to be effective-not very useful as many low income group use little sugar

Fortification of tea was seriously considered by the Food and Nutrition Board as a vehicle for iron fortification.

LIST OF SOME FORTIFIED FOODS AVAILABLE IN TRICHUR MARKET

<i>Food item</i>	<i>Nutrient fortified</i>	<i>Quantity</i>	<i>Price(Rs)</i>
Cornflakes(Kellogg's)	Vitamins,minerals	475g	125
Noodles(Maggi)	Ca and proteins	400g	38
Milk powder(Nido)	Vit A,C,E& Zn,Se	500g	150
Homogenized milk(Amul)	Vit A & D	2lit	27
Soya milk(Silk-USDA)	Ca &Vitamins	946ml	140
Biscuit(Marie gold)	Vitamins	1 packet	13
Orange Juice(Tropicana)	Vit A,C,E & Ca	200 ml	15
Salt (Anapurana,Sprinkle)	Iodine	1Kg	6-8
Oil (Saffola)	Vitamin E	1 lit	175

18. QUALITY CONTROL ASPECTS OF FORTIFICATION

Any fortification programme, be it addition of nutrients to the processed foods to improve their nutritional quality, restore their nutrients lost during processing or use of fortified foods as a public health strategy to control and

prevent deficiency disease in the community should be adequately protected by an appropriate legislation to ensure quality. Such legislation should cover the foods to be fortified, the purity of nutrients and other chemicals to be added and their levels in the fortified foods. Failure to implement and sustain affective quality system is likely to result in the failure of food fortification programmes. At the national level, overall responsibility of quality control of food fortification programmes should be clearly identified. Responsibility frequently rested in Ministry of health, food or agriculture.

The following steps are followed in the implementation of quality assurance programme for a fortified food product:

1. **Product specifications:** all specifications for the fortificant, vehicle and any other ingredient must be documented, including particle size, colour, potency, level of fortification and other requirements considered necessary. Acceptable deviations from specifications must be documented.
2. **Product safety assessment:** microbiological, chemical and physical hazards of all ingredients, as well as of the finished product must be assessed.
3. **Product analysis:** procedures for sampling and testing all ingredients, as well as the finished product must be documented.
4. **Determination of critical and quality control points:** the entire production process (including plant facility, equipment and environment) must be examined to identify stages (control points) at which inadequate quality control could affect product quality or lead to unacceptable health risks.
5. **Recall system:** a mechanism must be in place to recall products if necessary.
6. **Quality assurance audit:** periodic checks conducted to verify the quality assurance system is effective.

7. **Feedback mechanism:** mechanism to correct any product deficiencies identified by consumers
8. **Documentation:** all steps documented and readily available to relevant individuals and organizations.

Label declarations required for fortified foods

- 1) The claim 'fortified' shall be considered valid only when nutrient content analysis at any point in time within the shelf life of the product show at least 80% to 90% of the claimed fortification level depending on the nutrient analysed and the precision of the analytical methods used.
- 2) Fortification claims shall be based on the processed food as packaged and purchased by the consumers.
- 3) The fortification level shall be appropriately presented on the label indicating the following information
 - *Number of serving per container package*
 - *Serving size by weight or volume*
 - *Calories per serving (kcal)*
 - *Nutrients added and their corresponding amount expressed as % RDA per serving*
- 4) The terms "enriched", "added with", "supplemented with", and other similar terms shall be equivalent to the term "fortified". Descriptive terms like "rich in", "good source of" and "excellent source of" shall not necessarily imply fortification

19. LIMITATIONS

- Thorough knowledge of dietary habits and nutrient intakes in the target group
- A complementary educational programme is required particularly when the fortification influences organoleptical characteristic of food

- Not the ultimate solution of a nutritional deficiency problem but it may be an essential part of a nutritional programme

20. MISCONCEPTIONS

Mora (1995) quoted common myths about fortification that makes industry reluctant to adopt this technology, the main ones are

1. Food fortification technology is not fully developed
2. Food fortification technology is complex
3. Food fortification technology is expensive
4. Food fortification technology is a problem of legislation and enforcement
5. Food fortification technology is an economic burden

However, these apprehensions are not supported with any solid evidences. In fact, the present knowledge and experience have proved that food fortification is a simple, easy to adopt with minor modifications in the existing production plant. Technology for fortification of staple foods with micronutrients is not only well developed but has been successfully applied in industrialized and developing countries over several decades. Existing industry equipment could be used or otherwise acquired at relatively low cost. Percentage of added cost of fortification is relatively insignificant and could be passed on to the consumers. Appropriate legislation along with an industrial commitment can make food fortification successful.

CONCLUSION

Fortification of foods is currently used as a practical approach to improve the nutrient intake of individuals in the community. Food fortification campaigns in developing countries require careful planning, programming and communication. Strong development of a local food processing industry, the commitment and support of local governments through effective legislation and monitoring and a common culture that perceives gains from investing nominal amounts for personal health are the key factors in the long term success of fortification programme

“ No other technology offers as large an opportunity to improve lives.... at such a low cost and in such a short time” is the basis for World Bank policy on overcoming vitamin and mineral malnutrition in developing countries. The time is now ripe to use our knowledge, abilities and experience to restructure our action plans towards our efforts in eliminating micronutrient malnutrition.

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DISCUSSION

1. Why salt is not fortified with vitamin A?

Fortification of salt with vitamin a is not technically feasible, due to instability of vitamin A in salt. The hygroscopic nature of salt has prevented its use as a vehicle for Vitamin A fortification in countries of high humidity. In trying to overcome this problem, a new vitamin fortificant, encapsulated with limited success.

2. Name some fortified breakfast cereals

Corn flakes, Oats, Wheat flakes etc

3. How to reduce the loss of nutrients while cooking

Vitamin A: Avoid cooking at high temperatures - this destroys some of the vitamins by oxidation

B Vitamins: Avoid washing, soaking, or boiling, as B vitamins are water soluble and easily lost. Even brief boiling destroys up to a third of vitamin B12 and half the folic acid

Vitamin C: is water-soluble and heat sensitive. To reduce losses:

- Use as little water as possible
- Add food to rapidly boiling water
- Cover the pan
- Do not add sodium bicarbonate
- Avoid using copper pans
- Use cooking water for soups, sauces, and gravies
- Serve and eat promptly

Vitamin E: Avoid frying or baking as up to 50 % is lost

Proteins: Avoid overheating as this destroys some amino acids

Fats: Heating fats to high temperatures destroys essential fatty acids and can make them indigestible, and even possibly cancer inducing. Avoid reusing fat; frequent deep-frying; and frying for long periods

4. List some functions of salt

- It helps in the proper functioning of the nervous system,
- Salt is absolutely vital to making the structure of bones firm Salt is vital for sleep regulation
- Salt is vital for the prevention of gout and gouty arthritis
- Salt is most effective in stabilizing irregular heartbeats and, Contrary to the misconception that it causes high blood pressure, it is actually essential for the regulation of blood pressure - in conjunction with water.
- Salt is vital for balancing the sugar levels in the blood
- Salt is vital for the generation of hydroelectric energy in cells in the body. It is used for local power generation at the sites of energy need by the cells

5. Being costly, how will the fortified foods reach the vulnerable and low-income group people?

This can be achieved by providing the fortified foods through the public distribution system (PDS), midday meal programme in schools or through the Integrated Child Development Scheme (ICDS).

6 What is the RDA for iodine?

The daily requirement for iodine is reported to be 100-150 µg.

ABSTRACT

For the last two decades, micronutrient deficiencies specially anaemia, iodine deficiency disorders (IDD) and vitamin A deficiency have been a subject of concern for nutritionists and health authorities in developing countries. Among the available interventions for micronutrient malnutrition, food based approach has been considered as most acceptable safe, and sustainable. These food based strategies, include nutrition education, dietary diversification, dietary supplementation and food fortification (Subbulakshmi and Naik, 1999).

The advantages of food fortification relative to other modes of interventions have been widely noted, and one of this is that fortification programme can be implemented and yield results within a short period (Johnson *et al.*, 1988).

Food fortification has been defined as the addition of one or more essential nutrients in the food, whether or not it is normally contained in the food, for the purpose of preventing or correcting a demonstrated deficiency of one or more nutrients in the population or specific population groups (FAO/WHO, 1994). The food that carries the nutrient is referred as the food vehicle and nutrient added is the fortificant

The important aspects in fortifying programme include selecting an appropriate food item, the form of nutrients to be added, and ensuring the expected biological outcome of the programme (Rao, 2003)

Food fortification continues to be a widely used mechanism in many developed countries. In Japan, a multi nutrient enriched rice has been marketed since 1981 (Misaki and Yasumatsu, 1985). Dexter *et al* (1982) enriched durum wheat flour with vitamin mixture containing riboflavin, thiamine, and niacin for production of spaghetti. Breakfast cereals were fortified with vitamin and good storage stability was reported (Anderson *et al*, 1976). In Germany, a milk based fruit beverage fortified with calcium, phosphorous as well as vitamins has been marketed (Schauff, 1993).

In India fortification of cereal and cereal products with high protein source, minerals and vitamin were tried by the scientists of CFTRI (Subrahmanyam *et al.*, 1957). Fortification of bread with lysine, vitamins and minerals were opted by modern bakeries. Salt iodisation has been successful and used to prevent the iodine deficiency problems (Subbulakshmi and Naik, 1999). Many fortification programmes are still in experimental stage. Successive food fortification requires active collaboration between several sectors including the scientific communities, government agencies, private industry, consumer groups, and international organizations (Honeim *et al.*, 2001).

DIET AND CANCER

By

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(2004-24-01)**

**Ph.D Home Science
(Food Science and Nutrition)**

SEMINAR REPORT

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**DEPARTMENT OF HOME SCIENCE
COLLEGE OF HORTICULTURE
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2005**

DECLARATION

I, Aneena E. R (2004-24-01) hereby declare that the seminar entitled “**DIET AND CANCER**” has been prepared by me, after going through the various references cited here and has not been copied from my fellow students.

Vellanikkara



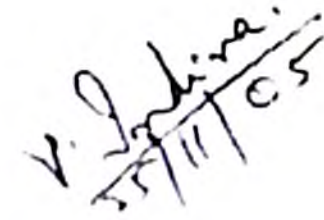
Aneena E R
(2004-24-01)

CERTIFICATE

This is to certify that the seminar report titled “DIET AND CANCER” has been solely prepared by Aneena, E.R (2004 – 24 – 01), under my guidance, and has not been copied from any seniors, juniors or fellow student’s seminar reports.

Vellanikkara

Date 25.11.05



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CONTENTS

SLNo	Title	Page No.
1	Introduction	1
2	Prevalance	2-5
3	Risk factors for cancer	5-6
4	Types of cancers	6-12
5	Macronutrients and cancer	12-14
6	Micronutrients and cancer	15-19
7	Carcinogens in foods	19-23
8	Anticarcinogens in foods	23-31
9	The cancer prevention diet	31-33
10	References	i-iii
11	Abstract	

DIET AND CANCER

INTRODUCTION

Cancer is one of the dreaded diseases and is the second most important cause of death in most affluent countries. The relationship between diet and health has been recognized throughout recorded history. Disease prevention through healthy preparation of foods and eating habits has been discussed for thousands of years. Since the 19th century, western scientific methodologies have been applied to the study of diet and disease with the intent of reducing the disease burden from non-communicable diseases (NCD) such as cancer, coronary heart disease (CHD), and other conditions endemic to societies after the advent of industrialization.

The idea that nutrition is an important factor in cancer causation is not new. Yong-He Yan living in Song Dynasty (960-1279 AD) thought that poor nutrition was a cause of the condition now known as oesophageal cancer. Lambe in 1815 warned against the danger of excess consumption of food in general and meat in particular. Roger Williams in 1908 observed that excessive feeding especially meat, deficient exercise and probably lack of sufficient vegetable food are the predisposing factors for cancer.

The role of diet takes special importance in countries like India which are fast moving towards industrialisation and westernisation. Dietary factors may be potentiating the role of other risk factors. Most of the cancers have some relationships with diet predominant among them are cancers of the upper aero digestive tract (mouth, throat), oesophagus (food pipe and lungs), stomach, large intestine, and breast cancer in women.

Cancer is a term used to refer malignant neoplasms and tumors. Neoplasia means cells in a tissue proliferate with out normal control on growth. Cancer is caused due to mutations or abnormal activation of cellular genes that control cell growth and cell mitosis (Sreelakshmi, 2000). The abnormal genes are called oncogenes. It may spread to other parts of the body through the lymphatic system.

or the blood stream. When these cells remain localized, it is called benign tumour and when it invades and spreads to other tissues of the body it is called a malignant tumour or in simple terms 'cancer'.

Under normal conditions, the growth, multiplication, repair and death of cells are biologically programmed. However gene mutations can lead to uncontrolled growth.

PREVALENCE OF CANCER

Malignant tumors are responsible for 12% of the nearly 5.6 million deaths world wide. In the year 2000, 5.3 million men and 4.7 million women developed a malignant tumor and altogether 6.2 million died of this disease. The prevalence of cancer in India is estimated to be 1.5-1.8 million.

In India, rates for oral and oesophageal cancers are some of the highest in the world (Gajalakshmi *et al.*, 2001). Cervical cancer is the most common cancer of the female genital tract in India. This accounts for almost 20% of all new cases diagnosed in the world annually (Ghim *et al.*, 2002). In India, the incidence of breast cancer increased by 50% between 1965 and 1985 (Saxena *et al.*, 2002)

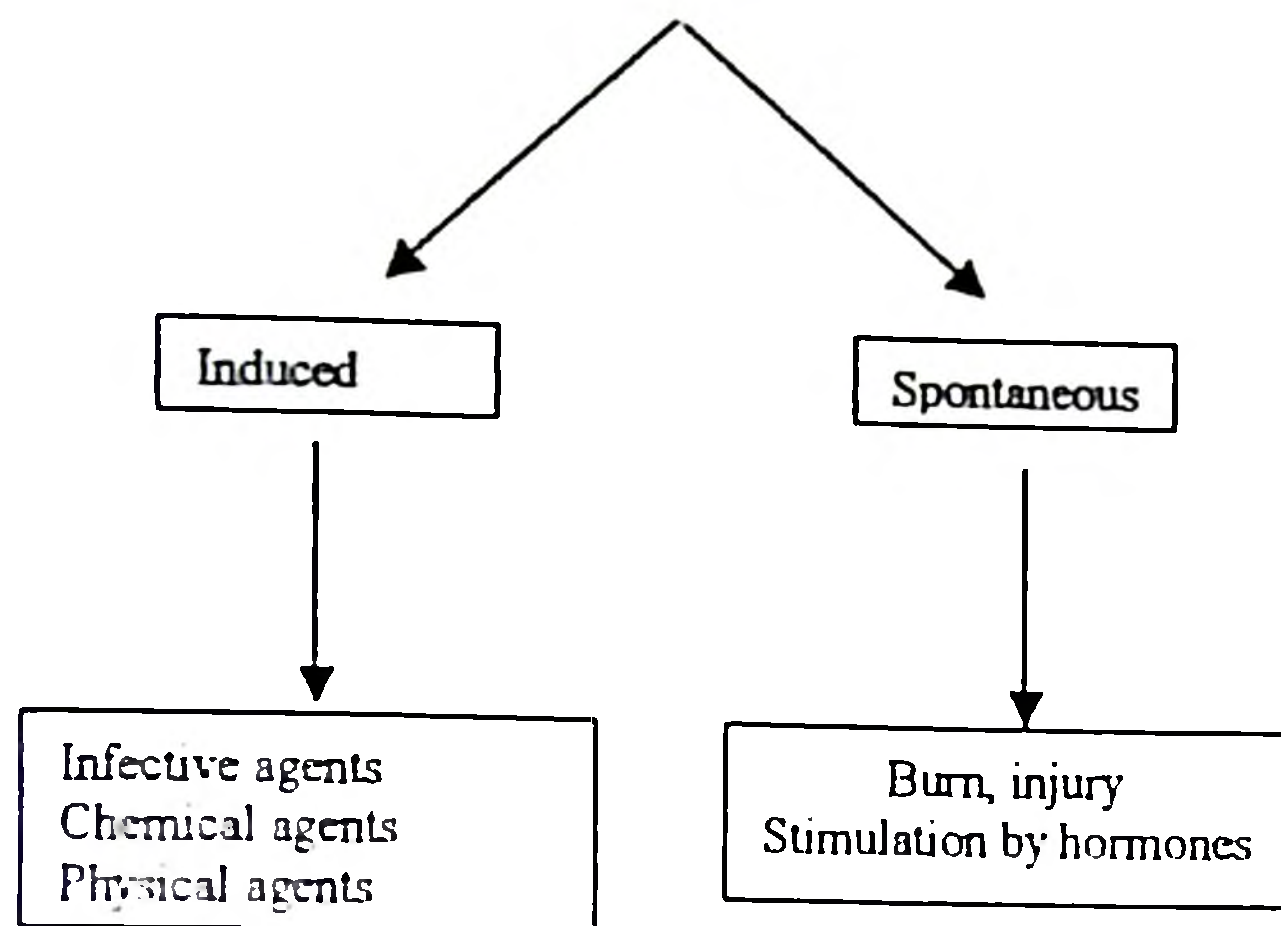
Global Incidence of Cancer by the Numbers

Lung cancer	1.2million
Breast cancer	1 million
Colorectal cancers	940,000
Stomach	870,000
Liver cancer	560,000
Cervical Cancer	470,000
Esophageal cancer	410,000
Bladder cancer	330,000
Leukemia	250,000
Prostate and testicular cancer	250,000
Pancreatic cancer	216,000
Ovarian cancer	190,000
Renal cancer	190,000
Endometrial cancer	188,000
Thyroid cancer	123,000

Cancer in Kerala

Population based cancer registries have provided reliable data on the occurrence of cancer in the State. The rate of cancer occurrence in Kerala and in India is much lower compared to western countries. It is estimated that 25,000 new cancer cases occur in Kerala in one year. Among males 50% of cancers in the mouth, throat and lungs are caused by Tobacco and alcohol habits. Among women tobacco related cancers are 15%.

Risk factors of cancer



These mutations can be spontaneous or induced. Spontaneous mutations occur following an injury e.g. burns, or prolonged stimulation by hormones. Mutations may be induced by infective agents like viruses, physical agents like radiations and by chemicals

In developing countries, up to 23 %of malignancies are caused by infectious agents E.g Hepatitis B and C virus (liver cancer), Human papilloma virus (cervical and ano- genital cancers), Helico bactor pylori (stomach cancer). Physical agents involves X -rays gamma rays, other ionizing radiations emitted by radio active substances

Chemicals can be classified as those naturally present in foods, formed during processing preparations and storage, and those added as food additives. Chemicals may be those which gain entry from outside through the food. These include nitrosamines, aromatic hydrocarbons, aromatic amines, and others. These substances attack the DNA and form complexes called DNA adducts. These act as ultimate carcinogen.

Under normal conditions the body has the mechanism to repair damaged DNA and an immune system which spots abnormal cells and destroys it. Binding of toxicants to plasma proteins and tissue proteins, protection by membrane barriers in the body, excretory process through urine, bile etc are some endogenous factors involved in the protection from toxic chemicals. Also there are several detoxifying enzyme system like the glutathione peroxidase, catalase, superoxide dismutase, phospholipids hydroperoxidase, glutathione-S- Transferase etc.

Stress

There is a relationship between the onset of cancer and emotional deprivation. Depression may affect the body's hormones and may inhibit the immune system's ability to counter disease.

Heredity

Family history of cancer plays a significant role in determining personal risk. Individuals of cancer-prone families need to pay particular attention to the avoidance of carcinogens and typically need closer cancer surveillance than other people.

Influence of Diet in Cancer

Dietary factors also play a significant role in cancer risk. At least one-third of annual cancer deaths in the US are due to dietary factors. A recent review on diet and cancer estimates that up to 80 percent of cancers of the large bowel, breast, and prostate are due to dietary factors. In January 1892, Scientific American printed the observation that "cancer is most frequent among those branches of the human race where carnivorous habits prevail" India is a developing country with most diverse populations and diets in the world.

Estimated percentages of cancer due to selected factors

Diet - 35-60%

Tobacco- 30%

Air and Water Pollution - 5%

Alcohol - 3%

Radiation - 3%

Medications - 2%

(Sinha *et al.*, 2003)

From the above data, it is evident that diet plays a significant role in predisposing cancer

Diet is an important factor in cancer aetiology because:-

- Diet may be a direct or indirect source of carcinogens.
- Carcinogens can be formed during storage, cooking, or preservation of foods
- Diet may contain inhibitors or anti promoters.
- Diet may modulate host mediated carcinogenic expression of xenobiotics through changes in nutritional status.
- Food stuffs may act as substrates for the formation of carcinogens in the body
- Food stuff may alter the bacterial flora of bowel leading to cancer.

TYPES OF CANCERS

Oral Cancers

Incidence rates for oral cancer in India are among the highest in the world (Fenley *et al.*, 2000). Most are associated with diet, weight, and other lifestyle factors. A significant lifestyle risk factor is betel chewing, a practice that is highly

prevalent in India. Betel quid contains a variety of ingredients such as lime, catechu, and areca nut and is often mixed with tobacco. A case-control study in Southern India investigated the influence of pan, body mass index (BMI), diet, infections, and sexual practices on oral cancer (Rajkumar *et al.*, 2003). BMI was inversely associated with oral cancer, and pan chewers with low BMI had a very high risk of developing oral cancer. Frequent consumption of fish, eggs, a variety of raw and cooked vegetables, and fruit was associated with a decreased risk of oral cancer. A study on reverse smoking (i.e. smoking with the glowing end inside the mouth) revealed that use of tobacco in this form conferred a 5.19 times higher risk of oral pre-cancerous lesions of the palate than did use of chewing tobacco. Diets low in vegetables and fruits and high in alcohol increase the risk of oral cancers.

Oesophageal cancer

In India, the incidence of oesophageal cancer is moderately high and is associated with certain diets and lifestyles. Oesophageal cancer is the second most common cancer among males and the fourth most common cancer among females according to combined data from cancer registries in India. Among risk factors for oesophageal cancer in India, betel quid chewing carries a relative risk of 1.5 to 3.5. Commonly used fresh and sun-dried vegetables and chillies also have a high content of nitrates or nitrosamines and may be associated with higher rates of oesophageal cancer. Various foods and food additives have been studied for their association with this disease. Spicy foods, chillies, betel chewing, tobacco chewing, smoking, alcohol etc are also responsible for this cancer.

Endometrial, Cervical, and Ovarian Cancers

Cancer of the female reproductive tract has a high incidence amongst Indian women. Human papilloma virus (HPV) is the most prevalent risk factor for cervical cancer and has been associated with cancer of the ovaries and

endometrium. Cervical cancer is the most common cancer of the female genital tract in India, with approximately 100,000 new cases occurring each year. This accounts for almost 20% of all new cases diagnosed in the world annually. A review of studies (Potischman and Brinton, 1996) on diet and cancers of the cervix, ovary, and endometrium have provided equivocal results. This study suggested that a diet high in carotenoids, vegetables, and fruits may reduce the risk of cervical, ovarian, and endometrial cancers; high intake of vitamins C and E may reduce the risk of cervical cancer; and a diet high in fish may reduce the risk of ovarian cancer.

Breast Cancer

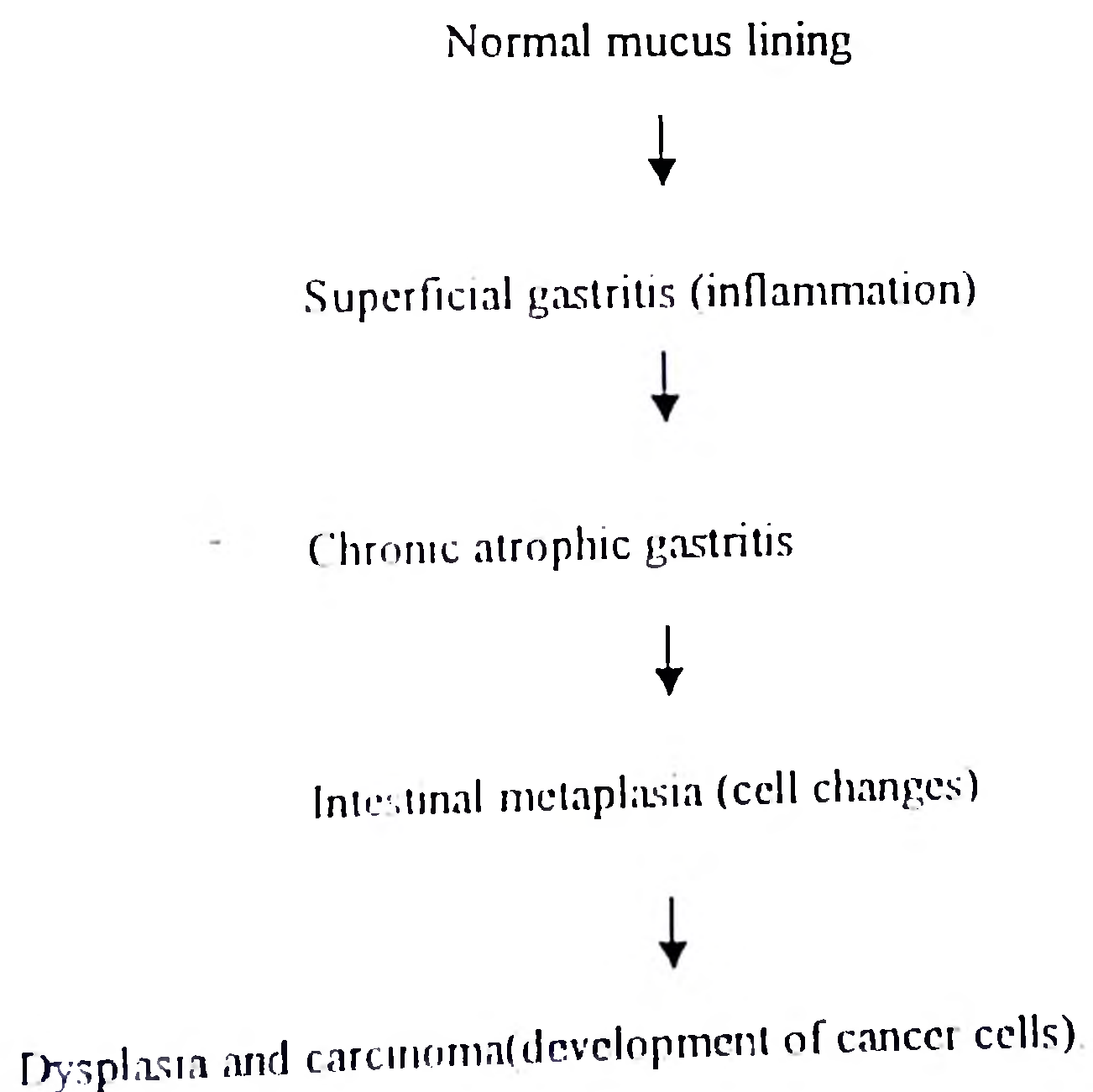
In India, the incidence of breast cancer is increasing, with an estimated 80,000 new cases diagnosed annually. The nutrition guidelines follow the World Cancer Research Fund recommendations that advocate having a diet containing vegetables and fruits in large amounts, reducing the intake of saturated fats, and increasing physical activity. Countries with a higher intake of fat, especially animal fat, have a higher incidence of breast cancer. In mammary tumor models, the tumor promoting effect of fat intake has been observed primarily for polyunsaturated fats when fed in the presence of high fat diets containing approximately 45% of energy.

Stomach Cancer

Compared to other countries, stomach cancer incidence rates are moderate to low in India, although certain populations, such as those in the Chennai area, have very high rates (Gajalakshmi *et al.*, 2001). A recent case-control study in Mumbai found that consumption of dried fish increased the risk while green tea consumption decreased the risk of having stomach cancer. A prospective case-control study from Trivandrum evaluated dietary risk factors for stomach cancer and found that high consumption of rice, spicy food, chili, and high-temperature

food increased the risk of developing stomach cancer(Mathew *et al.*, 2000). Fried foods are associated with higher rates of cancer due in part to the production of carcinogenic or mutagenic heterocyclic amines (HA) during the cooking process. It has identified that an increased risk of stomach cancer with moderate to heavy consumption of fried foods. This has been attributed to the presence of polycyclic aromatic hydrocarbons produced by the cooking method. The use of the spice turmeric is associated with a reduced risk of stomach cancer, in part because of its protective effect against the carcinogenic bacterium *H. pylori*, a major risk factor for stomach cancer.

- Stomach cancer seems to progress through certain stages,



Salt intake and gastric cancer

- High salt intake can induce atrophic gastritis which is a precursor to stomach cancer.

- Salt is considered a gastric tumor promoter. It promotes the carcinogenic effects of known gastric carcinogens.
- Elevated salt intake may potentiate *H. pylori*-associated carcinogenesis by inducing proliferation, cell hyperplasia, and glandular atrophy.
- Pickled vegetable and noodles are rich in sodium and low in vitamin C induces gastric cancer

Alcohol, Smoking and Gastric Cancer

Gastritis and reflux are established risk factors for gastric cancer. Cigarette smoking and alcohol consumption were considered to be risk promoters. Heavy drinkers are likely to be heavy smokers and cigarette use increases cancer risk and lowers immune status. Alcohol will destroy the gastric mucosa

Colon Cancer

High concentrations of carcinogenic bile salts, largely due to high fat diets, remaining for several days in colon, largely due to a low fibre diet, may have an irritating effect on the colon membrane. This may lead to the initiation or promotion of colon cancer. Dietary changes can reduce the risk of colon cancer. Burkitt (2000) observed that type and number of bacteria was influenced by the type of food eaten. This in turn greatly influences the stool volume. He found that high fat diet not only increased the amount of certain bile salts in the intestinal tract, but also increased the number of intestinal bacteria. They convert the bile salts into carcinogenic substances in colon. Diets high in fibre, not only create large stool volume, also it dilute the concentration of bile salts and carcinogens in the colon. It drastically reduces the transit time of the stool and thus the contact time of the carcinogens with colon membrane.

Pancreatic Cancer

A diet high in meat and fat may increase the risk of pancreatic cancer, while eating a lot of fruit and vegetables may reduce this risk. The pancreas produces pancreatic juice, which contains chemicals to break down fats and proteins in the diet. The pancreas' other important function is to produce two hormones, known as insulin and glucagon, which control blood sugar levels.

Prostate Cancer

Diet low in vegetables increase the risk of prostate cancer. High-fat diets alter the amounts of testosterone, estrogen, and other hormones in both men and women. A high fat diet that comprises mostly animal fat sources (such as dairy products, fatty meats and takeaway foods) may increase the risk. High red meat consumption is identified as a major cause. Lycopene - a potent antioxidant found in tomatoes, tomato-based products, watermelon and strawberries - may help to lower the risk of prostate cancer

Lung cancer

This is the leading cause of death from cancer in the world and smoking is mostly responsible. Diet low in vitamin A and carotene. Diets high in vegetables and fruits are protective against lung cancer.

Dietary factors in cancers –aetiology by site

Cancers	Dietary factors
Oesophagus	Alcohol, tobacco, low intake of fruits and vegetables
Stomach	Nitrates and nitrosamines, pickle and salted foods, smoked and fried foods, foods rich in starch with very little fruits and vegetables.
Colon and rectal	Refined carbohydrates, low <u>fiber</u> , low vegetable intake, high meat intake.
Nasopharyngeal	Salted fish
Larynx	Tobacco, alcohol
Lung	Tobacco, low intake of green and yellow vegetables
Bladder	Chemicals, artificial sweeteners, coffee.
Breast and cervical cancer	High intake of fat, low intake of fruits and vegetables.
Pancreas	Tobacco, coffee and meat
Liver	Mycotoxins and alcohol
Prostate	High meat intake and low <u>fiber</u> intake.

MACRONUTRIENTS AND CANCER

Calorie

An excess of calories in humans is associated with increased risk of cancer. Excess carbohydrates act as modifiers of carcinogens. On excess calorie intake, there is a high risk of endometrial and breast cancer. This is due to the

increased conversion of androgens into estrogens in adipose tissue. Obesity may possibly be associated with gall bladder cancer and colon cancer. Obesity and lack of physical activity are associated with increased risk at various cancer sites, including breast and endometrial cancer. Energy balance, which includes maintaining ideal weight through physical exercise, has been associated with decreased risk of breast cancer.

Proteins

High protein foods especially animal protein is a major source of mutagens. Protein foods when heated above 300°C leads to the formation of polycyclic aromatic amines. Polycyclic aromatic hydrocarbons are known to cause gastric cancer and cancers of alimentary canal.

Dietary fats

Diets high in saturated fats have been associated with increased risk for cancer (World Cancer Research Fund, 1997). Diets high in fat have been linked to increased risk of various cancers, particularly breast, colon, prostate, and pancreas, ovary, and endometrium. There is association between fat intake and the risk of breast and colon cancer is much stronger for total fat intake (Sinha *et al.*, 2003)

Diet high in fats has been linked to increased risk of various cancers, particularly breast, colon, prostate and possibly pancreas, ovary and endometrium. A high fat intake increases intestinal anaerobic bacteria, and biliary steroids secretion. Anaerobic bacteria are capable of synthesizing estrogens, which are believed to be potential carcinogens in mammary glands. Bile acids are degraded by intestinal micro flora to the secondary bile acids, deoxylithocholate, lithocholate, etc which may act as carcinogen in colon. According to Willett *et al*

(1992) there is increased risk of total saturated and monounsaturated fats are associated with increase in colon cancer.

Fatty foods affect the body in many ways and have a strong influence on hormonal activity in the body. First, high-fat diets increase the amount of estrogens, the female sex hormones, in the blood. It is known that many breast tumors are "fueled" by estrogens. Estrogens are normal and essential hormones for women and men, but the more estrogen there is, the greater the driving force behind some kinds of breast cancer. On high-fat diets, estrogen levels increase. When women adopt low-fat diets, their estrogen levels drop noticeably in a very short time. Vegetarians have significantly lower estrogen levels than non-vegetarians, in part because of the lower fat content of their diet.

Dietary fat intake and calorie intake is highly correlated. Dietary fat is the most concentrated source of energy. So reduction in fatty acid should also accompanied by reduction in calories and body weight.

Dietary fibre

Dietary fibre cannot be seen or taste but it can works wonders for our body. Dietary fibre is known as cancer fighter found only in the cell walls of plant foods. This protective effect is due to

- Fibers tendency to add bulk to the digestive system
- Shortening of the amount of time that wastes travel through colon
- Increased fiber decreases chances for intestinal walls get affected by toxic substances
- When bacteria in lower intestine break down fiber, butrate is produced which may inhibit growth of tumours of the colon and rectum
- High fiber diets are often lowers the absorption of fat
- Fibers bind with estrogen and prevents breast cancer

There are two types of fibres in our diet soluble fibre and insoluble fibre. Soluble fibre dissolves in water and is found in a variety of fruits, vegetables,

legumes, and grains. It cuts cholesterol, adds to the feeling of fullness, and slows the release of sugars from food in to the blood. Good sources of soluble fibres are oats, bran, apple, citrus fruits, straw berries, dried beans, rye flour, potatoes, raw cabbage etc.

Insoluble fibre does not dissolve in water and is found in grain bran, fruit pulp, and vegetable peels and skins. This type of fibre strongly linked to cancer protection and improved waste removal.

MICRONUTRIENTS AND CANCER

Besides the major nutrients there are some micronutrients which effectively protect our body. Micronutrients play a significant role in maintaining health and preventing disease, including cancer, through a wide range of mechanisms- anti-oxidation, anti-proliferation, and repair of DNA damage. Vitamin deficiencies, specifically of vitamins A, C and E, may contribute to the high prevalence of oral cancers in India (Tandon *et al.*, 2000). A study carried out in rural India found that the presence of lesions was associated in patients with oral pre-cancerous lesions with low plasma levels of vitamins E and b-carotene (Petel *et al.*, 2001). A study of Kurchias a tribal population in Kerala, India, who consume a diet high in micronutrients and have a low prevalence of CHD and other chronic diseases of aging, including cancer) found that levels of serum vitamins A and E were inversely related to levels of lipid peroxides and CHD risk factors (Reddy *et al.*, 1999)

Carotenoids\vitaminA

Ziegler (1989) examined the role of carotene rich foods and found that these foods reduce the risk of lung cancer.

Both are anticancer nutrients that support normal cell differentiation of tissues and internal linings. They prevent cancer cell formation by inhibiting the binding of

carcinogen to cell wall. Beta carotene may protect DNA in the nucleus of the cell by decreasing the bonding of chemicals to the membrane around the nucleus. Both nutrients are antioxidants and can scavenge free radicals.

Decreased levels of vitamin A are associated with increased rates of cancer of lungs especially and also of mouth, esophagus, bladder, cervix, and stomach. The major sources of beta-carotene are fruits and vegetables. Vegetables such as carrots, sweet potato, greens, spinach, and broccoli and the direct sources of vitamin A involves, non-vegetarian foods such as fish, eggs and liver.

Vitamin C

Vitamin C is involved in the cancer defense functions and it is highly protective against cancer.

Anti cancer aspects of vitamin c are listed below-

- Antioxidant activity
- Stimulates lymphocytes to produce interferon which decreases virus reproduction
- Supports thymus function, especially in strengthening and supporting antibody responsiveness
- Reduces production of nitrosamines from dietary nitrates and nitrites
- Reduces stomach, oesophageal and bladder cancers by means of its multiple protective effects on mucous membrane
- Along with folic acid vitamin C minimizes cervical dysplasia, and cancer.

Of a group of epidemiological studies in investigating the role of vitamin C, provides significant protection against cancer and tumerogenesis (Black, 1994). High maternal intake of vitamin C offers protection against childhood brain tumors (Krishnaswamy and Jagadeesan, 1999). Vitamin C is found in fruits

particularly citrus fruits and juices, and green leafy vegetables, sprouted pulses etc.

Vitamin E

Functions well with adequate levels of selenium as selenomethionine. This act as antioxidants and cell membrane protectors. It reduces the carcinogen production and strengthens immune cells and cell membranes against the penetration of viruses and toxic chemicals. Vitamin E has inhibited tumors in experimental animals and been linked to reduced risk of oral, stomach, and other cancer in epidemiological studies. In a study of effect of alpha tocopherol on lung cancer, among smokers, dietary intake of these nutrients from foods was found to be associated with a reduced risk for lung cancer (Blot *et al.*, 1993). Vitamin E has also been suggested to play a role in the prevention of nitrosamine induced upper gastrointestinal tract tumours by inhibiting the nitrosation of dietary amines in stomach

The major functions of vitamin E in cancer defense mechanism -:

- Vitamin E act as antioxidant and cell membrane protector
- Reduces the carcinogen production
- Strengthens immune cell and cell membranes against penetration of viruses and toxic chemicals
- Prevents nitrosamine formation

The major sources of tocopherol are unprocessed oils, animal fat, butter, egg yolk etc

Vitamin B₆

Vitamin B₆ is associated with endocrine metabolism, adrenal steroids, thyroid function, pancreatic function, thyroid and sex hormones and growth

hormone. It has a major role in endocrine metabolism, adrenal steroids, and sex hormones. Contributes to the function of over 100 enzymes in the body, the deficiencies are associated with cancer.

Vitamin B₁₂

The deficiency leads to pernicious anaemia. In pernicious anaemia, the stomach produces too little of a substance that it needs to take up vitamin B₁₂ from foods. This can also increase the risk of developing stomach cancer.

Vitamin D

Required for the mineral absorption in the body strongly protective against breast cancer and colon cancer. Vitamin D is essential for the prevention of skin cancer.

Minerals and Trace elements

Selenium

It helps to regulate the glutathione peroxidase, a strong antioxidant enzyme. Low selenium levels in the body are clearly associated with increased rates of leukemia and cancers of the breast, lungs, colon, rectum, prostate, ovary, skin, and pancreas. The major sources involve whole grains, legumes, and green leafy vegetables.

Zinc

Is another important mineral, which influences over 200 enzymes. It is an immune supporter and is important to the formation and function of many enzymes that work with detoxifying chemicals. Low levels are associated with

prostate, bronchial, esophageal, and colon cancers. Blood zinc levels have been reported to be inversely correlated with oral cancers.

Iron

Iron is a cofactor for many enzymes like catalase, peroxidase, which inhibit lipid peroxidation. Iron deficiency has been associated with cancers of upper alimentary tract which include oral cavity, oesophagus, and stomach. Iron deficiency is known to result in plumer-vinson syndrome, which in turn is associated with increased risk of cancers at above sites (Krishnaswamy and Jagadeesan, 1999). The increased risk of gastro intestinal tract tumors associated with iron deficiency in humans. It also revealed that there is increased risk of mammary tumors in iron deficiency.

Iodine

Iodine is essential micronutrient and thyroid cancers have been associated with endemic goiter. It is essential for the formation of Thyroid hormone. Thyroid hormone is essential for cell differentiation, induction of DNA and protein synthesis. Among 6-14-year-olds, goiter, caused by iodine deficiency and related to thyroid cancer, has a prevalence rate of 0.33 to 2.4%. (Chakravarty and Ghosh, 2000)

Calcium

Epidemiological studies indicate that high calcium intake reduces the risk of colon cancer by correcting irregular cells in colon.

CARCINOGENS IN FOODS

Carcinogens can be defined as substances that cause rapid uncontrolled division of cell

Trans fatty acids

They are the imitation of fats in shortenings and margarines and most commercial baked foods. They are commonly seen in all processed foods and snack foods. They increase the requirement of essential fatty acid. They competes with EFA in incorporation of total fatty acids, by altering the membrane bound enzymes and receptors. Trans fatty acids are strongly associated with cancer of lungs and reproductive organs.

Rancid fats-: Industrial processing and poor storage creates rancidity in vegetable oils. This rancid fats are a pool of free radicals and so known as potent carcinogens.

Polycyclic aromatic hydrocarbons

Cooking food at very high temperature, especially directly over the flame, generates chemicals which are carcinogenic in nature. Benzopyrene is produced by heat induced chemical reaction on fat. It is formed due to the incomplete combustion of oils and smoke which will get deposited on food. Cooking by direct smoking over the flame cooking over high temperature etc can lead to the formation of these carcinogens. All most all processed foods are prepared by these unhealthy cooking methods, so they are identified as the carriers of this carcinogen

Sodium nitrates

Nitrosamines are derived from the reaction of nitrate with a corresponding amine. They produce carcinogenic compounds nitrosamines and nitrosamides, which are carcinogenic. Of 300 nitroso compounds tested, 95% are found to be carcinogenic. It can be either preformed as in germinated barley, meat, meat products, salted fish etc. It can be formed from its precursors also e.g. green leafy

vegetables, spices, which are the sources of nitrates. Fresh vegetables, fruits, milk etc were found to be free from but it can be formed when these foods are grilled roasted, baked in an open air system. Nitroso compounds are formed during acidic conditions and chlorine acts as a catalyst. So pickles are one of the sources of these compounds. Cooking under high temperature and usage of open fire for cooking favours the formation of nitroso compounds. Alcoholic beverages contains nitroso compounds in higher levels. Hamburgers, hot dogs, such fast foods have become the most favorite food of youngsters today. All these foods contain cured and smoked meat and so contain high amounts of nitroso compounds.

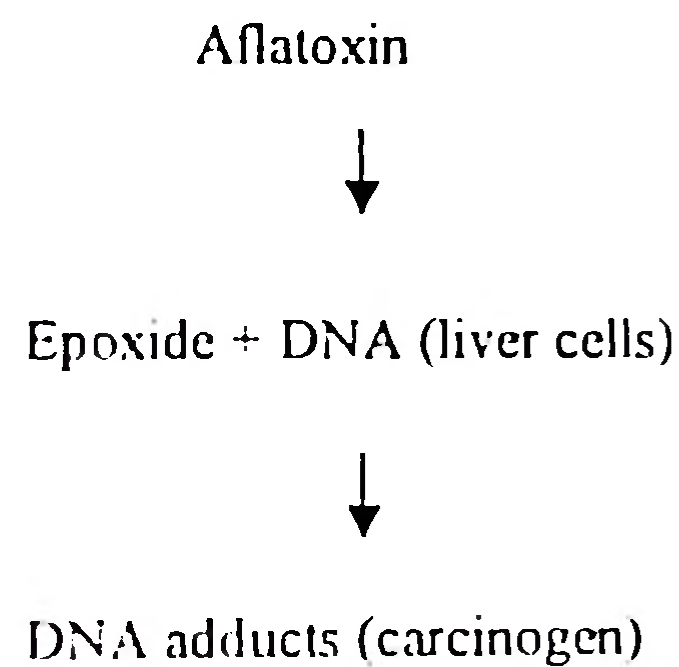
Food additives

Different food additives are identified as the sources of carcinogen. All non permitted food colours are mutagenic and most of them have been identified as potential carcinogens. Metanil yellow, a non permitted food colour was a common adulterant in foods like laddu, turmeric etc. Sudan dye and lead chromate are other non permitted food colours abundantly seen in foods and are known carcinogens. Permitted food colours also in high amounts will do harm. It caused different types of allergic reactions, damage to liver, kidney, etc that can lead to carcinogenicity. Jonnalagadda (2004) reports that processed meats, nonalcoholic beverages, miscellaneous ready to eat foods and sugar confectionaries etc contains food colours in highest concentrations of above 1000ppm while the allowed amount is 100ppm.

Monosodium glutamate (MSG) commonly called Ajinomotto is known to cause brain cancer. Artificial sweeteners, like saccharine, aspartame, cyclamates etc are also known potent carcinogens. Saccharine and cyclamate develop bladder cancer. Aspartame is associated with brain cancer. Refined carbohydrates like sugar, high fructose corn syrup, white flour are devoid of nutrients. Refined carbohydrates provide too much calories and can lead to obesity and cancer.

Mycotoxins

Aflatoxins have been received considerable attention and have linked with liver cancers. They are toxins produced by infestation with moulds. Aflatoxin contamination affects liver. The major plant foods which are contaminated are ground nuts, corn, coconut, and pepper which are stored under improper conditions of temperature and humidity. Aflatoxins on oxidation in liver forms epotoxins which affects the DNA of liver cells and forms DNA adducts which induces cancer in the cells.



Tobacco and cancer

Tobacco chewing and smoking are the 2 important risk factors of oral cancer in India. Lung cancer risk of regular smokers to non smokers is between 20-30 fold. More than 50% of cancer in men and 20% in women are related to tobacco. Tobacco related cancer is in the oral cavity, pharynx, larynx, and esophagus.

Alcohol and cancer

- Causes hepatic injury and cirrhosis and lead to the formation of hepatomas.

- Alcohol can act as a co carcinogen, promoter and facilitates transport of other carcinogens.
- Alcohol induce enzymes which bioactivates carcinogens.
- Produce nutrient deficiencies.
- Induce immuno suppression which enhance carcinogens

ANTI CARCINOGENS IN FOOD

Phytochemicals are fascinating yet mysterious group of thousands of chemicals found in plant foods. They possess several physiological functions. Phytochemicals are essentially of plant origin. They act as powerful antioxidant protecting cells and organs from free radicals, neutralising their damage effects (Polasa, 1998).

The major anti carcinogens in foods are:-

- Isothiocyanates
- Lycopene
- Flavanoids
- Bioflavanoids
- D -limonene
- Phyto oestrogens
- Protein inhibitors
- Phytic acids
- Saponins
- Phenolic compounds

Isothiocyanates

- They prevents carcinogen from activation.

- Alcohol can act as a co carcinogen, promoter and facilitates transport of other carcinogens.
- Alcohol induce enzymes which bioactivates carcinogens.
- Produce nutrient deficiencies.
- Induce immuno suppression which enhance carcinogens

ANTI CARCINOGENS IN FOOD

Phytochemicals are fascinating yet mysterious group of thousands of chemicals found in plant foods. They possess several physiological functions. Phytochemicals are essentially of plant origin. They act as powerful antioxidant protecting cells and organs from free radicals, neutralising their damage effects (Polasa, 1998).

The major anti carcinogens in foods are:-

- Isothiocyanates
- Lycopene
- Flavanoids
- Bioflavanoids
- D –limonene
- Phyto oestrogens
- Protein inhibitors
- Phytic acids
- Saponins
- Phenolic compounds

Isothiocyanates

- They prevents carcinogen from activation.

- They counteract the poisonous effects of carcinogens that have been activated.
- They speed up their removal from body (Arab and steck, 2000).
- Effective in fighting lung and oesophagal cancers.

Isothiocyanates can be seen in cruciferous or cabbage family vegetables such as broccoli, cauliflower, turnips, cabbage, radish, horseradish etc.

Lycopene

Is a part of carotenoid family, it is a pigment present in most of fruits and vegetables. The mechanism of action involves:-

- Act as a free radical fighting antioxidant. So that it they cannot attach to the cells and disintegrate the immune system.
- Potential nitrosamine inhibiting agent.

Studies show that lycopene helps to prevent prostate, lung and stomach cancers (Giovannucci, 1999)

Flavanoids

Comprise one of the largest groups of secondary plant metabolites. More than 400 flavanoids have been identified of which some are flavonols, flavones, flavanones, catechines, biflavins. Most of these flavanoids are structurally polyphenols having antioxidant activity. Flavanoids and tannins are similar to some other antioxidants such as ascorbic acid and tocopherol and this antioxidant effect is attributable to the radical scavenging activity of flavanoids.

The major functions are the following:-

- Structurally flavanoids having anti oxidant activity

- Blocks heterocyclic aromatic amines for promoting gastric and colorectal carcinogenesis (Susanta, 2001).
- Important in protecting DNA from high rates of mutation

Quantitative distribution of flavanols in some vegetables (mg/100g fresh weight)

Vegetable	Quercetin	Kaempferol	Myricetin	Lectrolin
Lettuce	0.7-3.0	0.2	0.1	0.1
Onion	28.4-48.6	0.2	0.1	0.1
Redpepper	0.1	0.2	0.05	0.7-1.4
Broad bean	2.0	0.2	2.6	0.1

(Kallo and Singh 2001)

Bioflavanoids

The important bioflavanoids are Kaemperol, quercetin and myricetin

- They are complex compounds which are closely associated with vitamin C.
- They increase the effectiveness of vitamin C, a recognised potent antioxidant
- Quercetin has anticarcinogenic activity.
- Quercetin inhibits growth of several types of cancer cell proliferation.

D-limonine

- This is a terpenoid and is a major component of citrus fruit peel.
- It is a flavouring agent.
- Induces the enzyme, glutathione-s-transferase.

Phytoestrogens

Induces isoflavones and lignins, which are found in cereals and pulses including sorghum, millets and particularly soyabean. Lignins are anti proliferative and abnormal cell growth inhibitor. Lignins are found in whole grain products, seeds, fruits and berries. They are weakly estrogenic and competes with steroid hormones for various enzymes. They also stimulate production of sex hormone binding globulin in the liver.

Protein inhibitors

They are widely distributed in cereals and pulses like barley, wheat, oats, rye, soyabeans, kidney beans, chick peas and other pulses. They inhibit the action of protease, enzyme which is believed to contribute to the invasive capacity of some cancer cells

Phenolic compounds

They include ellagic acid found in fruits and nuts like straw berries, raspberries, blackberries, walnuts. They inhibit nitrosation reaction by trapping nitrate to form n-nitrophenolic compound

Phytic acid

Found high amounts in sesame seeds, limabeans, peanuts, and soyabean and also in cereals, nuts, seeds, and pulses. It controls cell proliferation.

Glucosinolates

Found in cruciferous vegetables is the most important intra cellular anti oxidants protecting the cells against oxygen free radicals (Franceschi, 1991).

Saponins

Saponins are found abundantly in soyabean (5% of dry weight). They exhibit cytotoxic effects and growth inhibition against a variety of cells. They bind to bile acids and regulate their recirculation. They have known mutagenic inhibiting activity.

Classes of potentially anticarcinogenic phyto chemicals

Phytochemical classes	Food source
Carotenoids	Yellow/orange vegetables, dark green vegetables
Dithiolthiones Glucosinolates/Indoles Isothiocyanates	Cruciferous vegetables
Allium compounds	Onions, garlic, leeks, chieives
Coumarins	Vegetables and citrus fruits
Flavanoids	Most vegetables and fruits
Plant sterols	Vegetables
Isoflavanoids	Soyabeans

Allium vegetables and Cancer

Vegetable belonging to allium family like garlic, posses strong antioxidant activity. The purported health benefits include chemoprotective, antibiotic, and cholesterol lowering properties. Garlic compounds have been shown to inhibit tumorigenesis thus reducing the risk of cancer (Saxsena, 1998)

Spices and cancer prevention

Ginger

- Known to produce substances that protect stomach lining
- Prevents the formation of gastric ulcers.
- Stimulate gastric activity, and stimulates the bowel to empty quickly.
- Promotes digestion

- Deactivates Carcinogens.
- Enhance tissue levels of protective enzymes.

Turmeric

Among the most studied in recent years is turmeric, an ingredient in the common Indian curry and a spice that has been shown to be a potent antioxidant and anti-inflammatory agent with additional promise as a chemo-preventive agent. In a study in human blood cancer cell lines, turmeric suppressed and destroyed blood cancer cells. Turmeric has been shown to suppress tumour initiation, promotion, and metastasis in experimental studies. To illustrate, turmeric may block the activity of nuclear factor kappa-B (NfκB), which, in an activated state, appears to be associated with cancer cell growth in many cell types. Turmeric also has been found to inhibit the growth of 19 clinical strains of *Helicobacter pylori*, a carcinogenic bacterium linked to the increased risk of adenocarcinoma of the stomach and colorectal adenomas (Aggarwal, 2003).

Other spices

Cumin seeds and basil leaves -Significantly decreased the incidence of both Squamous cell carcinoma and hepatomas. Cumin seeds contain Pthalides which increases anti cancer protective enzymes.

Poppy seeds- significantly inhibited Benzo [a]Pyrene-induced Squamous Cell Carcinoma

Cloves- contain eugenol which have anti mutagenic effect

Antioxidants in Foods

Apart from nutritive value of vegetables, some other constituents which play a vital role in disease prevention are antioxidants (Singh and Nirmal, 2001). In human terms, lipid peroxidation can be directly linked to ageing, cancer, heart diseases and other regenerative diseases. Vegetables are an important and cheaper source of protective nutrients such as vitamins and minerals. Some vitamins which act as antioxidants are Vitamin E, Vitamin C and Beta Carotene.

Their presence in some common food stuffs are listed below.

Food stuffs	Vit E (mg)	Vit C (mg)	Beta carotene (mg)
Apple (1 medium)	0.44	8	0.04
Broccoli	1.32	116	1.30
Brown rice	4.00	0	0.00
Brussels sprouts	1.33	96	0.67
Carrot (1 medium)	0.28	7	12.00
Cauliflower	0.05	54	0.01
Chick peas	0.57	2	0.02
Corn	0.15	10	0.22
Grapefruit (pink, 1/2)	0.31	47	0.19
Navy beans	4.10	2	0.00
Orange (1 medium)	0.31	75	0.16
Orange juice			
Pineapple	0.22	124	0.30
Soybeans	0.16	24	0.02
Fresh spinach	3.35	3	0.01
Strawberries	0.57	16	2.30
Sweet potato	0.23	84	0.02
(1 medium with skin)	0.32	28	15.00

Broccoli

Broccoli is known as the crown jewel of nutrition for its vitamin and fibre richness and low in calorie. Multiple cancer fighting properties owing to vitamin C, beta carotene and fiber. It is rich in phytochemicals- indole carbinol and sulphoraphane. Indole carbinol breaks down estrogen. A medium sized stalk of broccoli provides 220% of the daily value of vitamin C, 15% of the daily value of vitamin A, in the form of beta carotene.

Soyabean and cancer prevention

Soya foods prevent cancer as well as diseases associated with estrogen deficiency. The isoflavonoid genistein is a phytoestrogen found in abundance in soya food. In soyabean oil the active antioxidant is tocopherol. Other antioxidant components are isoflavine, glycosides, phospholipids, phenolic acids.

THE CANCER PREVENTION DIET

Some dietary changes have a preventive effect for many types of cancer. Boosting your intake of vitamin-rich vegetables and fruits, for example, strengthens your immune system and helps knock out cancer cells.

It has recommended that low fat, high fibre and micronutrient rich diet as cancer prevention diet. Lower fat intake to about 20% of total calories or a maximum of 50 grams of fat per day is suggested. More fat should be mono and poly unsaturated fats with reduction of saturated fat intake and little or no consumption of refined or heated oils. Complex carbohydrates can come up to 60% of the diet. Increase in dietary fibre help to improve the functioning of gastro intestinal tract. Protein intake should be 15% of the diet. Frequent usage of fried, grilled, salted and smoked foods should be avoided. Animal foods cooked at a high temperature also should be avoided. Vegetable protein is beneficial in cancer

prevention than animal protein. The usage of alcohol, coffee, preservatives in foods, excess salts etc should be discouraged. For cancer prevention, the NIN recommends a diet that includes high intake of fresh vegetables and fruits, with spices such as turmeric, in adequate amounts (Krishnaswamy and Polasa 1995). The overall best foods for cancer prevention include organic white meats of poultry, and fish, whole grains, vegetables, and fruits.

Whole grains and cancer prevention

They are concentrated sources of dietary fibre resistant starch and oligosaccharides, fermentable carbohydrates etc. Whole grains are rich in antioxidants trace elements and phenolic compounds. They are significant sources of phytoestrogens. They mediate glucose response.

Tips for Increasing Fibre in Your Diet

- Whenever possible, do not remove the fibre-rich peels and skins of fruits and vegetables. Just be sure to wash them thoroughly before eating.
- Choose products that are minimally processed, like whole-wheat bread instead of white bread and brown rice instead of white rice.
- Plan each of your meals to include whole grains, fruits, vegetables, and legumes.
- To avoid intestinal discomfort when increasing fibre intake, it is best to increase gradually and drink plenty of water.
- Snack on baby carrots, apples, strawberries, oranges, and other fibre-rich fruits and vegetables.
- Top your breakfast cereals with dried fruits like raisins or dates, or fresh fruits like strawberries or peaches.

Tips for Increasing Phytochemicals in Your Diet

- Use fresh or frozen vegetables instead of dried or canned.
- Keep cooking to a minimum. Most phytochemicals do not fare well when exposed to high temperatures.
- Add fresh garlic (not garlic powder) to almost any meal. Or if a recipe already calls for garlic, just add more than it recommends.
- Eat whole, not refined, grains. Phytochemicals are found in the highest concentrations in the nutrient-rich fibre that coats the starchy center of the grain. During processing, this fibre is usually lost.
- Eat a variety of vegetables. Try something new at least once a week and continue to incorporate the new vegetables into your meals.

Some dietary suggestions for cancer prevention

- Avoid obesity
- Cut down total fat intake
- Eat more fibre foods
- Include cruciferous vegetables
- Include foods rich in vitamin A and C
- Avoid alcohol consumption
- Lower intake of processed foods
- A variety of foods should be used in moderately

We had a predominantly plant based diet and with the advent of western life style we are moving towards a diet rich in animal proteins. This coupled with other habits like smoking and alcohol will lead to increase in the chronic disease burden especially cancer and cardiovascular diseases. Prompt action has to be taken to spread the message of healthy life style and dietary practices. If prevention is best to cure, nutrition is the safest option.

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DISCUSSION

1. Do heavy metals cause cancer? In which foods they occur?

Heavy metals like cadmium, lead, aluminium etc will cause cancers. This is many due to industrial pollution. These metals can get in to our food, by food additives, packagings etc. It has reported that ayurvedic drugs contains of heavy metals in higher levels.

2. Will permitted colours cause cancer?

Yes. Permitted colours above safe levels are mutagenic.

3. Which age group is more vulnerable to cancer?

Cancer is a disease, which can affect anybody at ant time in our life span. It has no relationship with age or sex.

4. Which type of cancer is more prevalent in Kerala?

Oral and oesophagal cancer is most prevalent in Kerala.

5. Will artificial sweetener cause cancer?

Artificial sweeteners above safe limits will cause cancer.

6. What is the safe level of Mono sodium glutamate?

There is no specified safe level for MSG. Symptoms of intoxication tends to occur within one hour after eating three grams or more of MSG on an empty stomach or without other food. Generally low levels are considered as safe. On long term usage it will cause cancer.

Abstract

Cancer is one of the most dreaded diseases and is the second most important cause of death in the affluent countries. Cancer is the term used to refer malignant neoplasms and tumors. Neoplasia means cells in a tissue proliferate with out normal control on growth. Cancer is caused by activation of cellular genes that controls the cell growth and cell mitosis.

The prevalence of cancer in India is estimated to be 1.5-1.8 million. Cancer of oral cavity is an important contributor to cancer morbidity and mortality in India. Cancer of female reproductive tract has a high incidence among Indian women.

Diet is an important factor in cancer aetiology and cancer prevention. Diet is a complex composite of nutrients and non-nutrient food constituents and many nutrients have relationship with the formation of cancers. Excess calorie intake can lead to obesity and cancer incidence. Higher the body weight, higher is the risk of colorectal, breast, prostate, and ovarian cancers. High intake of fat is associated with increased incidence of cancers. Breast cancer, a highly prevalent cancer in India may be due to high fat intake and low fibre content in diet and obesity. High fat intake and low fibre diet also induces colo-rectal cancer. The major risk factor for gastric cancer can be identified as high consumption of fried foods, spicy foods and hot beverages.

Other constituents in foods like dietary fibre, selenium, zinc, iron, beta-carotene, vitamin C, vitamin E etc have significant protective function against cancer. Isothiocyanates present in cruciferous vegetables like broccoli, cauliflower, turnips etc have been proved to be effective against lung cancer and oesophagal cancers. Lycopene, an important antioxidant, and the major colouring pigment in tomato helps to prevent prostate, lung and stomach cancers. Phytochemicals present in plant foods act as powerful antioxidants protecting

cells and organs from damage caused by the free radicals, neutralizing their damaged effects.

Carcinogens in foods include trans fatty acids, rancid fats, poly hydroxy aromatic amines, nitrates and nitrosamines, artificial sweeteners, food preservatives etc. Some of these are produced during cooking, processing, and storage of foods. Unhealthy habits like tobacco chewing, smoking, and alcoholism, which are widely prevalent in India remains as the predisposing factors for different types of cancers.

The consumption of fruits and vegetables in the daily diet contribute various micronutrients, which have many biological effects. The non-nutrient components of these protective foods are also as important as micronutrients. Expected benefits of incorporating a wide variety of fruits and vegetables whole grains liberally in the routine diet may not only lower the risk of cancer but also reduce the risk of other chronic diseases.

NUTRITIONAL ANAEMIA AND IT'S CONSEQUENCES

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(2004-16-03)

M. Sc. Home Science
(Food Science and Nutrition)

SEMINAR REPORT

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DECLARATION

I, Remyamol, K.K (2004 - 16 - 03) here by declare that this seminar report titled "NUTRITIONAL ANAEMIA AND IT'S CONSEQUENCES" have been prepared by me independently, after going through the various references cited herein and has not been copied or adopted from any of the fellow students or previous seminar reports

ellanikkara

date: 15/10/05


Remyamol, K.K
(2004 - 16 - 03)

CERTIFICATE

This is to certify that the seminar report titled "NUTRITIONAL ANAEMIA AND IT'S CONSEQUENCES" has been solely prepared by Ms. Remyamol, K.K (2004 - 16 - 03), under my guidance, and has not been copied from any seniors, juniors or fellow student's seminar reports.

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CONTENTS

Sl.No.	Title	Page No.
1	Introduction	1
2	Definition of anaemia	2
3	Types of anaemia	3
4	Causes of anaemia	3
5	Iron deficiency anaemia	4
6	Megaloblastic anaemia	10
7	Functional consequences of anaemia	15
8	Anaemia in pregnancy	16
9	Prevention of anaemias	18
10	Conclusion	22
11	Discussion	23
12	References	i-ii

NUTRITIONAL ANAEMIA AND ITS CONSEQUENCES

INTRODUCTION

Anaemia is a major global problem affecting 20-70 per cent of the population in various countries. In India, it is an important public health problem affecting people from all walks of life. The disease is of particular significance in preschool and school children and pregnant women because of the high prevalence (50-70%) and the adverse functional consequences. In school children anaemia impairs scholastic performance and in young women, the reproductive performance. Anaemia is also directly and indirectly responsible for 10-20 per cent of maternal deaths, high incidence of premature births and intrauterine malnutrition.

Iron deficiency related health problems are widely prevalent in India, particularly among the poorer sections of people. IDA is reported to be prevalent in 50-80 per cent of the population in different parts of the country (Kochupillai, 1997). The department of women and child welfare has reported that 47 per cent prevalence of moderate to severe anaemia (Hb < 9g%) among pregnant mother in India.

The ICMR estimates indicates that nearly 35-50 per cent of the children in India in rural areas and belonging to low income group have anaemia that is commonly due to iron deficiency. Earlier studies from the National Institute of Nutrition (NIN), Hyderabad and other studies showed an average anaemia prevalence rate of 68 per cent in preschool children (Damodaran *et al.*, 1979). According to WHO, the average community prevalence of anaemia in India is reported to be around 50 per cent, as higher prevalence of 70-80 per cent is among women and pre-school children. Even among adult men, prevalence of anaemia ranges from 30-40 per cent.

In India, the national level prevalence of anaemia amongst pregnant women was reported to be 87 per cent (ICMR, 1989). A subsequent study by ICMR in 5 states of India reported wide geographical variation within the country - the lowest prevalence of 33 per cent reported from Andhrapradesh, 62.5 per cent

in Delhi and 98 per cent in Rajasthan (ICMR, 2001). It is estimated that in India at least 13 million women during pregnancy suffer from anaemia (MOHFW, 1998).

In addition to pregnant women and adolescents, young children of 6-24 months who are growing rapidly also form another group at the risk of iron deficiency anaemia. A prevalence rate of over 65 per cent in preschool children has been reported in various studies undertaken in rural and urban India (Seshadri *et al.*, 1994).

National and regional surveys indicate that the prevalence of anaemia could be as high as 74 per cent in children below 3 years of age, 85 per cent in expectant mother and 60-70 per cent among adolescent girls and 30 per cent among males (MOHFW, 1998).

DEFINITION OF ANAEMIA

Anaemia is defined as reduction in the haemoglobin (Hb) level in circulation (Raman and Sarma, 1998). Nutritional Anaemia may be defined as the condition that results from the inability of the erythropoietic tissue to maintain a normal haemoglobin concentration on account of inadequate supply of one or more nutrients leading to reduction in the total circulating haemoglobin (Sreelakshmi, 2003).

Cut-Off points for haemoglobin values for diagnosis of anaemia (WHO, 1972)

Group	Haemoglobin (g/dl)
Adult men	<12
Adult women	<12
Pregnant women	<11
Lactating women	<12
Children till 6 years	<11
Other children	<12

TYPES

Hypochromic and microcytic anaemia

If there is an insufficiency of iron for the formation of haemoglobin, the red blood corpuscles are pale and small and the anaemia is said to be hypochromic and microcytic.

Normocytic normochronic anaemia

This is due to acute blood loss or haemolytic anaemia or bone marrow failure.

Megaloblastic anaemia

Vitamin B₁₂ and folic acid are co-enzymes in the DNA synthetic pathway. A deficiency of the vitamins or impairment in their utilization results in damaged or inadequate synthesis of DNA. The synthesis of RNA and protein is unaffected so there is cytoplasmic enlargement, not matched by DNA synthesis which appears to delay or block mitotic division. Thus there appears to be asynchronism between cytoplasmic maturation and nuclear maturation. If the maturation of the red blood corpuscles in the bone marrow is impaired by lack of folate or Vitamin B₁₂, the cells which enter the blood stream are irregular in size and shape, but usually larger than normal, and contain their full complement of haemoglobin. This anaemia is also known as orthochromic macrocytic.

Dimorphic

If both iron and either folate or Vitamin B₁₂ are deficient it gives rise to hypochromic macrocytic or dimorphic anaemia.

Multi-factorial causes of anaemia

Immediate causes

- Deficient nutrition intake
 - Iron (Bio available)
 - Folate
 - Vitamin B complex
 - Vitamin C
 - Protein

- **Interfering factors**
 - Phytates
 - Tea
 - Coffee
- Conditions such as respiratory infection and diarrhea
- Increased blood loss with inadequate iron intake.

Underlying causes:

- Inadequate diet in quantity/quality
- Poor environment
 - Water
 - Sanitation
 - Food hygiene
- Poor healthcare, eg. no immunization
- Excessive menstruation, child birth, malaria, parasitism, hook worm, Schistosomiasis and trauma

Basic causes:

- Lack of awareness of food values
- Inadequate food security
- Inadequate carrying capacity
- Poverty
- Inadequate health policy/programme

IRON DEFICIENCY ANAEMIA

Iron deficiency anaemia is one of the most common nutritional disorders world-wide, especially in India and other developing countries. Young children and women in the reproductive age group are the most vulnerable to iron deficiency anaemia. Survey in different parts of the country reveals that 87 per

cent of pregnant women suffer from anaemia and about 10 per cent have severe anaemia (H <80/l)

Iron deficiency anaemia refers to anaemia that results from iron deficiency or impaired iron status. Iron dependent physiologic functions can become impaired before anemia develops. Although iron deficiency anaemia can occur in all socio-economic groups, it has historically been most common among the poor as is still the case today.

Aetiology:

- Inadequate iron intake secondary to a poor diet such as vegetarian life style with insufficient haem iron. The average cereal legume based diets as consumed in most developing countries would appear adequate in iron content (20-22 mg) for an adult. But the availability of iron from such diets is very poor. Only 3-5 per cent of dietary iron is absorbed in normal apparently healthy individual (Rao, 1978)
- Blood losses can occur in accidental haemorrhage, in chronic diseases such as tuberculosis, ulcers or intestinal disorders, or excessive blood donation or due to hook worm infestation.
- In rural areas, post partum haemorrhage on account of poor obstetric practices pregnancies and prolonged periods of lactation depletes iron stores with each successive pregnancy and this is reflected in the high incidence of anaemia with high parity.
- Resurgence of anaemia is another important factor resulting in high incidence of anaemia. In women, using intrauterine contraceptive device, menorrhagia (increased blood loss) may result in further depletion of already poor stores of iron.
- Deficiency of iron in diet during periods of accelerated demands like infancy (rapid expanding blood volume), adolescence (rapid growth & onset of menses in girls) and pregnancy and lactation can result in anaemia - Losses of iron may occur due to excessive sweating in tropical climate.
- Inadequate absorption of iron can occur in diarrhea, (sprue and pellagra) or when there is lack of acid secretion by the stomach or in chronic renal diseases when

antacid therapy is given. Gastrectomy impairs non absorption by decreasing HCl and transit time through the duodinum.

- Excessive amount of phytates and phosphates in the diet and excess consumption of tea can decrease the absorption of iron (Dokkum, 1992).

Other non-nutritional causes:

- a) Haemoglobinopathies due to abnormal formation of haemoglobin as in thalassaemia, sickle cell anaemia and others. In thalassaemia there is a failure of protoporphyrin synthesis.
- b) Bone marrow hypoplasia or aplasia due to drug idiosyncrasy, radiation therapy, antitumour and anticonvulsant drugs.
- c) Anaemia of chronic disorder due to increased destruction and infiltration of cells as in malignancy, leukaemia, hypersplenism.
- d) Toxins like lead also inhibit synthesis of haem and globin.
- e) In anaemia of chronic renal failure, there is a decrease in erythropoietin synthesis.
- f) Infection, rheumatoid arthritis, systemic lupus erythematosus and Crohn's diseases. In chronic inflammation and malignancy, there is lack of iron release from macrophages to serum, reduced red cell life span and inadequate erythropoietin response.

Diagnosis

Haemoglobin measurement:

It is important tool in diagnosing anaemia. Haemoglobin can be measured from finger prick blood samples by direct colour comparison of acid or alkaline haematin where a calorimeter is not available or calorimetrically as a cyanmethaemoglobin or oxyhaemoglobin. Method based on direct colour comparison are not recommended, even from a field area. It is advisable to bring the blood samples on a filter-paper and estimate haemoglobin colorimetrically after extraction.

Most patients develop symptoms of anaemia when the haemoglobin level is approximately 8-11 g/dl.

The plasma ferritin level

It provides a measure of iron stores

Transfusion saturation

It can be used as a gauge of iron supply to the tissues. It is calculated by dividing serum iron by total iron binding capacity. In deficiency levels falls from a normal level of 30 per cent to 16 per cent.

Reticulocyte counts

In anaemia the count increases from the normal value of 0.5-2.0 per cent. Very high value (76%) indicates severe anaemia.

The ratio of zinc protoporphyrin to haeme is a sensitive indicator of iron supply to the developing red blood cells.

When insufficient substrate iron is available to incorporate into porphyrin, zinc is then substituted. Although it can combine with globin and circulate, this zinc containing molecule cannot bind oxygen.

By itself haemoglobin concentration is unsuitable as a diagnostic tool in cases of suspected iron deficiency anaemia for three reasons

- 1) It is affected only late in the diseases
- 2) It cannot distinguish iron deficiency from other anaemias
- 3) Haemoglobin values in normal individuals vary widely

Stages of iron deficiency:

There are three stages of iron deficiency been described

- a) First stage is characterized by decreased storage of iron without any other detectable abnormalities
- b) An intermediate stage of latent iron deficiency that is iron stores are exhausted, but anaemia has not occurred as yet. Its recognition depends upon measurements of serum ferritin levels. The percentage saturation of transfer in falls from a normal value of 30 per cent to less than 15 per cent. This stage is most widely prevalent stage in India.

- c) The third stage is that of overt iron deficiency when there is a decrease in the concentration of circulating haemoglobin due to impaired haemoglobin synthesis.

In case of selecting indicators for detecting iron deficiency anaemia in the community, the filter paper method was found to be the most suitable to carry out haemoglobin estimation in field conditions although haemocue provides accurate results.

Clinical findings

The end results of iron deficiency is nutritional anaemia which is not a disease entity. It is rather a syndrome caused by malnutrition in its widest sense. Besides anaemia there may be other functional disturbances such as impaired cell mediated immunity reduced resistance to infection, increased morbidity and mortality and diminished work performance

An iron deficiency anaemia becomes more severe, defects arise in the structure and functions of the epithelial tissues, especially of the tongue, nails, mouth and stomach. The skin may appear pale and the inside of the lower eyelid may be light pink instead of red. Fingernails can become thin and flat and eventually koilonychia (spoon shaped nails) develops. Mouth changes include atrophy of the lingual papillae, burning redness and in severe cases a completely smooth waxy and glistening appearance to the tongue (glossitis). Angular stomatitis and dysphagia may occur. Gastritis occurs frequently and may result in achlorhydria. Progressive, untreated anaemia results in cardiovascular and respiratory changes that can eventually lead to cardiac failure

Chronic long term iron deficiency symptoms reflect a malfunction of a variety of body systems. The general symptoms are lassitude, fatigue, breathlessness on exertion, palpitations, dizziness, tinnitus, headache, dimness of vision, insomnia, paraesthesia in fingers and toes and angina.

Inadequate muscle function is reflected in decreased work performance and exercise tolerance. Neurologic involvement is manifested by behavioural changes, such as fatigue, anorexia and pica especially pagophagia (ice eating). Abnormal cognitive development in children suggests the presence of iron

deficiency before it has developed into overt anaemia. Growth abnormalities, epithelial disorders and a reduction in gastric acidity are common. A possible sign of early iron deficiency is reduced immune competence, particularly defects in cell-mediated immunity and the phagocytic activity of neutrophils, which may lead to an increased propensity for infection.

Neurological and behavioural implications

Some of the manifestations of anaemia are fatigue, dullness, lack of concentration, reduced activity all leading to poor performance.

Iron deficient young adolescents have been shown to score relatively lower in test of academic performance. Also they have been found to be more disruptive, irritable and restless in the class room. Iron deficiency is also sometimes associated with pica, temper tantrum and breath holding spells by children.

Treatment

Treatment should focus primarily on the underlying disease or situation leading to the anaemia. Oral administration of inorganic iron in the ferrous form - ferrous sulphate 50-200 mg (60 mg elemental iron) 3 times daily for adults and 6 mg/kg for children. Other salts absorbed at about same degree are ferrous forms of lactate, fumarate, glycine, sulphate, glutamate and gluconate. Ferric hydroxide poly maltose complex has proved to be a suitable effective and safe alternative to oral salts in the treatment of iron deficiency anaemia by both oral and parenteral routes.

Iron is best absorbed when the stomach is empty. Under these conditions, it leads to cause gastric irritation. Gastrointestinal side effects of nausea, epigastric discomfort and distention, heartburn, diarrhea or constipation can be minimized by increasing the dose slowly over a few days until the required dosage is reached and by giving the iron in divided doses atleast three times per day. Use of chelated form of iron can result in improved absorption and can reduce the likelihood of gastrointestinal distress. Ascorbic acid greatly increases iron absorption through its capacity to maintain iron in the reduced state.

Iron therapy should be continued for several months even after restoration of normal haemoglobin levels, to allow for repletion of body iron stores.

In iron deficiency anaemia when dietary folate is also low, treatment with iron alone precipitate folate deficiency because more is needed for production of erythrocytes and the supply of folate becomes insufficient.

MEGALOBLASTIC ANAEMIA

Haemopoietic tissue is one of the number of rapidly proliferating tissues in which DNA synthesis is intense. Both Vitamin B₁₂ and folate are essential for DNA synthesis and deficiency of either or both causes a failure of DNA synthesis and disordered cell proliferation. Haemopoiesis is particularly susceptible and division of cells, is delayed and eventually halted. Morphological changes appear in the marrow cells. In the erythrocyte series there changes are described as megaloblastic because the cells appear abnormally large. They are nucleated red cell precursors. Megaloblast appear in bone marrow as well as in peripheral blood.

Vitamin B₁₂ deficiency

Vitamin B₁₂ must be bound to intrinsic factor, produced by the parietal cells of the stomach, before it is absorbed in the terminal ileum. Inability to produce intrinsic factor results in pernicious anaemia. The red cell count is often less than 2.5 million and a large proportion of the cells are macrocytic. The anemia occurs chiefly in middle aged and elderly persons and may be a genetic defect. Antibodies against gastric mucosa can probably be responsible for destroying the mechanism of producing intrinsic factor. The disease thus arises as an autoimmune disorder.

Nutritional vitamin B₁₂ deficiency is not very common, even in populations where intake of the Vitamin B₁₂ is far below the daily requirement of 0.5µg - 1µg. Vitamin B₁₂ deficiency takes atleast 3 years to appear (Vitamin B₁₂ is stored in the liver where there may be upto 3 years supply).

Causes of pernicious anaemia

Inadequate ingestion

A poor diet lacking in microorganisms and animal foods which are the sole source of Vitamin B₁₂. Chronic alcoholism, poverty, religious taboos and dietary facts can cause B₁₂ deficiency. Infants solely breastfed for long period of time develop anaemia due to deficiency Vitamin B₁₂ deficiency.

Inadequate absorption and utilization

In adequate or absence of secretion of intrinsic factor due to heredity or congenital production of defective intrinsic factor gastric atrophy, endocrine disorders associated with gastric damage or due to gastrectomy. Bacterial proliferation in stragnt loops and in tropical sprue there is malabsorption of B₁₂. Parasitic infestation such as fish tapeworm may remove Vitamin B₁₂ from the gut

Inadequate utilization

This is due to the presence of Vitamin B₁₂ antagonists

Increased requirements

Hypothyroidism increases haematopoiesis. During infancy and pregnancy there is increased requirement of Vitamin B₁₂ as it is essential for nucleic acid synthesis

Clinical features:

Patients with pernicious anaemia have a lemon yellow or pale skin. Anorexia, glossitis, achlorhydria, abnormal discomfort, frequent diarrhea, weight loss and general weakness can also occur. The surface of the tongue is usually smooth and atrophic but sometimes it is red and inflammated. Gastric secretions are devoid of pepsin, acid and intrinsic factor. Numbness of limbs coldness of extremities and difficulty in walking are manifestations of neurologic changes. The haemoglobin content may be as low as 8 per cent. Demyelination of white fibres of spinal cord occurs in severe cases. Psychiatric symptoms may occur associated with low levels of Vitamin B₁₂ in the plasma, but in the absence of

other signs of neuropathy. Pernicious anaemia is associated with an increased risk of gastric cancer. Clinical manifestations are mental apathy, pigmentation, growth retardation and megaloblastic bone marrow. In young females there may be infertility.

Diagnosis:

The differential diagnosis of pernicious anaemia from other forms of megaloblastic anaemia depends upon the age of the patient (common among females aged between 46 and 65), the finding of histamine fast or pentagastrin - fast achlorhydria, upon the absence of pregnancy and the lack of evidence of malnutrition, malabsorption or structural change in the small intestine. Plasma Vitamin B₁₂ is below 160 mg/L while plasma folate is usually normal.

Treatment:

If the haemoglobin level is under 4 g/dl blood transfusion should always be given. Physical activity should be at a minimum until the haemoglobin is above 7 g/dl.

Hydroxocobalamin should be given in a dosage of 1,000 µg intramuscularly twice during the first week, then 250 µg weekly until the blood count is normal. Then 1,000 µg every six weeks is given. Folic acid should never be used alone in the treatment of pernicious anaemia as it does not prevent the development of neurological complications and may precipitate them.

Within 48 hours of the first injection of a cobalamin the bone marrow shows a striking change from a megaloblastic to normoblastic state. Within two or three days the reticulocyte count begins to rise, reaching a maximum about the 4th or 7th day.

To cope up with regeneration of blood, ferrous sulphate 200 mg three daily is given.

Vitamin B₁₂ levels in maternal milk and serum are low in pernicious anaemia. The clinical picture gets corrected by administering a single dose of 50 µg of Vitamin B₁₂ to the mother or to the infant.

Dietary consideration:

Poor appetite and gastrointestinal discomfort seriously interfere with an adequate food intake so that the patients often present a picture of general nutritional deficiency. A high protein diet of 100 to 150 g of protein with high calorie diet is recommended. Supplementation of liver extracts would be effective in the treatment of pernicious anaemia.

Achlorhydria retards digestion, hence fat in the diet should be kept to moderate levels, restricting especially fried foods that may further delay gastric emptying.

A soft or clear liquid diet is preferable until glossitis completely disappears. Spicy food should be avoided. A soft diet is recommended since there is anorexia and irritation of gastrointestinal tract.

Supplementation with ascorbic acid is essential if citrus fruits and other rich sources of vitamin C are not ingested. High protein, high calorie beverages, two or three times daily should be given.

FOLATE DEFICIENCY

The main source of folate are green leafy vegetables, liver and eggs. The daily recommended allowances vary from 25 mcg to 400 mcg per day for infant to pregnant women.

Causes:**Poor dietary intake**

Megaloblastic anaemia is common among poor vegetarians. This is due to poor intake of milk, fresh fruits and vegetables. As such the Indian diet is a poor source of folic acid and cooking practices commonly encountered tend to destroy the folic acid to a considerable extent.

Low absorption

This anaemia in babies is more frequent in those born to mothers who also have a folic acid deficiency. Anaemia is present in infants whose diet lacks in Vitamin C because folic acid cannot be converted to its active form folic acid.

Folate absorption is impaired in pregnancy. Vitamin B₁₂ deficiency can result in folic acid deficiency by causing foliate entrapment in the metabolically useless form of 5 methyl tetrahydrofolate.

Increased requirement

Increased requirements due to growth and pregnancy are believed to be the most important causes. This increased need arises from the increase in the maternal blood volume, growth of the uterus, placenta and foetus. (Baumslag *et al.*, 1985)

Infestation and infection

Malarial infection play a part in pathogenesis of megaloblastic anaemia. Chronic infestation and parasitic infestation may impair absorption of folate

Drugs

Anticonvulsant drugs and oral contraceptives may impair foliate absorption in some women. Loss of folic acid occur in haemodialysis. Patients taking anticonvulsant drugs for treatment of epilepsy tend to become folate deficient

Alcoholics comprise the only group that generally has all metabolic defects leading to folic acid deficiency. Less utilization and increased excretion, increased requirement and increased destruction of folic acid results in deficiency state in alcoholics

Clinical Features:

- Glossitis is less common
- Neurological problems are very rare
- Haemoglobin levels may be as low as 4 g/dl

Treatment:

- Folic acid in dose of 5 to 10 mg daily is effective
- Patients who have less than 5 g/dl of haemoglobin need blood transfusion

Dietary consideration:

Food rich in folic acid like pulses, green leafy vegetables, cluster beans, ladies finger, gingelly seeds, liver and eggs should be included on the diet.

FUNCTIONAL CONSEQUENCES OF ANAEMIA

1) Neurological and behavioural implications

Some of the manifestations of anemia are fatigue, dullness, lack of concentration, reduced activity, all leading to poor performance.

Iron-deficient young adolescents have been shown to score relatively lower in tests of academic performance. Also they have been found to be more disruptive, irritable and restless in the classroom. Iron deficiency is also associated with pica, temper tantrums and breathholding spells by children. Iron deficiency anaemia is also lead to irreversible impairment of child's learning ability and other behavioural abnormalities (Seshadri and Gopaldas, 1989).

2) Gastro intestinal manifestations

Iron deficiency is associated with varying gastrointestinal manifestation. Epithelial changes including metaplasia of buccal and oesophageal mucosa are seen. Tissues of iron deficient subjects contain reduced cytochrome and other iron requiring enzymes. Intestinal absorption has been shown to be reduced especially for xylose and fat.

3) Immunocompetence and infection

Iron is essential for normal development and function of lymphoid tissue. Free iron is essential for multiplication of all bacteria except lactobacillus. Iron deficiency results in a reduction, of cell mediated immunity. These changes in immune response are seen early in iron depletion. On rehabilitation improvement in immunocompetence precedes increase in haemoglobin concentration.

4) Change in epithelial cells

In untreated megaloblastic anaemia epithelial cell changes similar to those seen in erythroblasts of bone marrow occur. These include an increase in the size of both nuclear and cytoplasm with multiple nuclei (Ployploidy). These changes are seen as buccal mucosa and tongue (manifesting as glossitis) urinary tract (increased incidence of urinary infection) and uterine cervix resembling dysplasia. In jejunum and large intestine villi are shorter, sometimes atrophid and manifest as malabsorption and diarrhea.

5) Reproductive outcome

During pregnancy, iron deficiency along with deficiency of folate and vitamin B₁₂ result in poor foetal growth, prematurity and intrauterine death of foetus due to severe anaemia. The foetus is bone with poor stores of iron and folate at birth and suffers from anaemia from early infancy due to poor availability of iron and folate from breast milk

6) Poor work capacity

The endurance capacity of the anaemias was significantly lower than normal subjects (Srijaya and Jhansi Rani,2003). Non-anaemics expended less energy than anaemics for the same activity (Devadas,1988). In adult men or women, anaemia results in poor work output since the work capacity is reduced considerably due to muscle fatigue. Also lack of concentration in the work results in mistakes, sometimes fatal one

ANAEMIA IN PREGNANCY

In pregnancy, anaemia has a significant impact on health of foetus as well as that of mother. Anaemia especially if severe, may impair the oxygen delivery to placenta and foetus and interfere in normal intrauterine growth (Thangaleela and Vijayalakshmi,1994). In maternal anaemia placental weight, volume and surface area were reduced (Agarwal, 1991). Maternal anaemia resulted in 12 to 28% foetal loss, 30% of prenatal deaths and 7 to 10% neonatal death. The remaining births have around fifty percentage chance of resulting in a

low birth weight baby. Low birth weight is the most important factor in determining the chance of survival of the new born.

Anaemia in pregnancy is also associated with increased maternal morbidity. Maternal deaths to the extent of 15-20% are directly or indirectly due to anaemia (Thangaleela and Vijayalakshmi, 1994).

Low availability and poor absorption of iron and repeated and closely spaced pregnancies place a constant drain on the iron stores of pregnant women. Epidemiological studies have demonstrated significant relationships between serum vitamin A level and biochemical indicators of iron status. Vitamin A is involved in the regulation of iron transport from the liver and it also improves the haemoglobin level by impairing the absorption or utilization of iron by bone marrow. Studies have shown a beneficial effect of Vitamin A supplementation on the haemoglobin values of pregnant women (Paul *et al.*, 1994).

<u>Severity of anaemia</u>	<u>Cut-off level of haemoglobin</u>
Mild	- 9 g/dl and above but below 11 g/dl
Moderate	- 7 g/dl and above but below 10 g/dl
Severe	- Below 7 g/dl

(WHO, 1989)

Any pregnant women whose haemoglobin falls below 11 g/dl is considered to be anaemic. Studies carried out at NIN on pregnant women belonging to lower socio-economic group showed that prevalence of anaemia was between 50 and 90% in the third trimester of pregnancy.

A significant fall in birth weight due to increase in prematurity rate and intrauterine growth retardation has been reported to occur when maternal haemoglobin level falls below 8 g/dl. Anaemic pregnant women are prone to urinary tract infection. The effect of anaemia and urinary tract infection in pregnancy could also be the cause of low birth weight babies.

Causes

- Maternal dietary inadequacy
- Consequent under nutrition

Risk-care approach

All the pregnant women whose haemoglobin is below 8 g/dl should be considered as risk category. Anaemic pregnant women with haemoglobin level less than 8 g/dl should be given special care during pregnancy to improve haemoglobin either by oral or by parenteral iron therapy depending on the period of gestation.

Low availability and poor absorption of iron and repeated and closely spaced pregnancies plays a constant drain on the iron stores of pregnant women (Paul *et al*., 1994) Hence it is recommended that regular supplementation of iron for expectant mothers should be emphasized and nutrition education regarding the consumption of balanced diet should form a component of any effort which aims at improving the overall nutritional status of the expectant mothers

PREVENTION OF ANAEMIAS

Prevention of nutrition-related anaemia depends on adequate dietary intake of iron, vitamin B₁₂ and folate as well as the full complement of other essential nutrients. Folate deficiency anaemia usually occurs among women late in the course of pregnancy, among small premature infants and among alcoholics. Strict vegetarians who consume no foods of animal origin, especially women who are pregnant or nursing should take supplementary source of vitamin B₁₂

The national goals and approaches to controlling IDA

The National Tenth Plan has set the goal of reducing the prevalence of anaemia by 25 per cent among children and pregnant and lactating women. National programmes and institutional approaches are being undertaken to achieve this goal

The major approaches to controlling IDA, which are not mutually exclusive, are medicinal supplementation with iron and folic acid and food based approaches, i.e., dietary diversification and fortification of foods

Supplementation

To prevent anaemia under National Nutritional Anaemia Prophylaxis Programme contribution of iron and folate tablets to pregnant women during last trimester and for pre-school children is in operation as a part of MCH services.

The National Nutritional Anaemia Prophylaxis Programme in India (NNAP Programme)

This programme is in existence since 1970 aims at significantly decreasing the prevalence and incidence of anaemia in women in the reproductive age group, especially pregnant women. Their policy emphasize the following strategies.

- Promotion of regular consumption of food rich in iron.
- Provision of iron and folate supplementation the form of tablets to the high risk groups
- Identification and treatment of severely anaemic cases.

The suggested prophylactic doses for pregnant women is 60 mg elemental iron + 500 µg folic acid daily for at least 100 days during pregnancy and preschool children receive 20 mg elemental iron + 100 µg folic acid daily at least 100 days

In addition to increased iron and folate intake improvement in environmental sanitation and personal hygiene are also needed to control worm infestation and infections. Deworming done regularly would help in reducing the incidence of anaemia and improve the efficacy of iron supplements

Government organizes "Matra Suraksha Abhiyan" (Mother protection campaign), free tablets of iron and calcium are given to pregnant women. This free service by the government helps poor women to improve their anaemic conditions

Food fortification with iron to control anaemia

In view of widespread iron deficiency and also ineffective distribution of iron-folate tablets through the anaemia prophylaxis programme due to logistic

reasons, it is important to diversify and improve the availability of iron in the diet. Also fortification of foods with iron would act as a long-term measure to improve the iron balance in the entire population. Fortification has been successful in mitigating the problems of nutritional anaemia to a large extent in rural and even tribal areas (Saxena, 1996).

Food fortification is the process by which a nutrient is added to commonly eaten foods to improve the quality of the diet. *Fortification* is the addition of nutrients at levels higher than those found in the original or in comparable foods. The food that carries the nutrient is referred to as the *food vehicle*, and the nutrient added is the *fortificant*. Salt is considered as an eminently suitable vehicle for iron fortification in India as it satisfies all the criteria for an ideal vehicle. Salt is consumed in India by all segments of population, rich as well as poor perhaps more by the poor (Rao, 1991). Two different technologies of fortification of common salt were developed at the NIN, Hyderabad as a long-term strategy to control the population (Nair *et al.*, 1998). These are,

- **Iron fortified salt** - common salt fortified with iron
- **Double fortified salt** - common salt fortified with iron iodine

Salt consumption lies within a narrow range of 12-20 g/day with an average intake of 15 g/day/person. Of the estimated annual production of 7 million tons of salt about 4.5 million tons are estimated to be available for human consumption.

Iron fortification of wheat flour and bread is currently employed in the developing countries for the prevention of iron deficiency. Fortification of cereal and grain products is a relatively inexpensive and effective means of increasing iron intake. The absorption of iron from fortified cereals can be increased three fold if the cereals are also fortified with about 5 mg of vitamin C/mg of iron.

So effort should be made to manufacture double fortified salt in large scale and made the same available in the market to everyone so that people can buy and consume the same to eliminate the problems of iron deficiency among the

general population and specially the children (Vijayalakshmi and Padmapriya, 2003).

Dietary diversification

Dietary diversification helps to improve absorption of iron by lowering inhibitors and increasing promoter concentrations. Addition of vitamin C to a meal will help to increase the bioavailability of iron. Vegetables and fruits which are important sources of vitamin C enhances iron absorption. Green leafy vegetables had low total iron content but higher bioavailable iron (NIN, 1995). This may need nutrition education and changes in dietary habits of the population.

Nutrition Education

Emphasis should be laid on educating both the health functionaries as well as the general population about anemia. All medical, health and social workers horticulture department and voluntary organizations have role to play in promoting the consumption of iron rich foods. Following points need to be considered for promotion of the strategy,

- (1) Promotion of consumption of pulses, green leafy vegetables, other vegetables and meat products. To get vitamin B₁₂ consumption of meat or eggs need to be encouraged. Encouraging the use of iron acquired foods during processing like rice, flakes and dry fruits
- (2) Creation of awareness in mothers, attending antenatal clinics, immunization sessions, anganwadi centres and crèches about the prevalence of anaemic, ill effects of anaemia and its preventable nature
- (3) Addition of iron rich foods to the weaning of food in infants
- (4) Regular consumption of food rich in vitamin C such as oranges, guava, amla etc. to promote iron absorption
- (5) Promotion of home gardening to increase the availability of common iron rich in food such as green leafy vegetables
- (6) Encouraging the use of iron pans and iron ladles

- (7) Periodical administration of antihelminthic drugs.
- (8) Discouraging the consumption of foods and beverages like tea and tamarind that inhibit iron absorption especially by the vulnerable groups like pregnant women and children.

CONCLUSION

Anaemia is ignored in most developing countries even though it is one of the most prevalent public health problems and has serious consequences for national development.

Anaemia should be addressed through health facilities and existing programmes that work directly with community and community workers, such as water and sanitation or environmental health or hygiene and infectious diseases control programme

Agriculture extension or food security programme can help increase production and consumption of food rich in iron and other micronutrients. The private sector can also help, especially food and pharmaceutical manufacturers, marketers and distributors. The two most important nutrients viz. iron and folic acid can be easily provided by the inclusion of the cheapest of green leafy vegetables which luckily grow throughout in India.

DISCUSSION

1. What is achlorhydria?

Achlorhydria is defined as failure of the intragastric pH to fall to less than 4 under maximal stimulation.

2. How copper is related to anaemia?

Copper is not part of the haemoglobin molecule but aids in its synthesis by influencing the absorption of iron, its release from the liver on its incorporation into haemoglobin molecule.

3. Is there any ill effects caused by excess intake of iron?

Yes. Excess intake of iron may cause gastro intestinal side effects of nausea, epigastric discomfort and distention, heart burn, diarrhea or constipation.

4. What is fortification?

It is the process by which a nutrient is added to commonly eaten foods to improve the quality of the diet. *Fortification* is the addition of nutrients at levels higher than those found in the original or in comparable foods. The food that carries the nutrient is referred to as the *food vehicle*, and the nutrient added is the *fortificant*.

5. How can vegetarians improve their diet, in case of B₁₂ deficiency anaemia?

The only source of vitamin B₁₂ is the animal origin. In vegetarians diet the availability of vitamin B₁₂ is increased by using fermented foods.

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ABSTRACT

Nutritional anaemia is defined as the condition that results from the inability of the erythropoietic tissue to maintain a normal haemoglobin concentration on account of inadequate supply of one or more nutrients leading to the reduction in the total circulating haemoglobin (Sreelakshmi, 2003). It may be due to dietary deficiency of iron, folate, vitamin B₁₂, vitamin B₆, vitamin C, protein, vitamin E and copper.

Among nutritional anaemias Iron Deficiency Anaemia (IDA) is the most common nutritional disorder world-wide, especially in India and other developing countries. Young children and women in the reproductive age group are most vulnerable to Iron Deficiency Anaemia (Seshadri *et al.*, 1994). These anaemias are due to reduced intake, impaired absorption, increased loss and demand of nutrients

National and Regional surveys indicate that the prevalence of anaemia could be as high as 74 per cent in children below 3 years of age, 85 per cent in expectant mother and 69-70 per cent among adolescent girls and 30 per cent among males (MOHFW, 1998). Anaemia in pregnancy is associated with increased maternal morbidity. Maternal death to the extent of 15-20 per cent are directly or indirectly due to anaemia (Thangaleela and Vijayalakshmi, 1994).

The general symptoms associated with anaemia include fatigue, breathlessness on exertion, palpitation, dimness of vision, paraesthesia in fingers and toes and diminished work performance. The clinical symptoms include glossitis and koilonychia (Sreelakshmi, 2003).

BIOAVAILABILITY OF DIETARY IRON

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**(2004-16-02)
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SEMINAR REPORT

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DECLARATION

I, Jyothi,P (2004-16-02) hereby declare that the seminar entitle "Bioavailability of Dietary Iron" have been prepared by me, after going throug various references cited at the end and has not been copied from any of my fellow students.

Vellanikkara

Date 15-10-2005


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2004-16-02

CERTIFICATE

This is to certify that the seminar report titled "BIOAVAILABILITY OF DIETARY IRON" has been solely prepared by Ms. Jyothi.P (2004-16-02), under my guidance, and has not been copied from any seniors, juniors or fellow students seminar reports.

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CONTENTS

SI.NO	TITLE	PAGE NO
1.	Introduction	5
2.	Distribution of iron in the body	7
3.	Functions of iron in the body	7
4.	Effects of iron deficiency	7
5.	Signs and symptoms of Iron deficiency	8
6.	Iron deficiency and its causes	8
7.	Iron requirements	10
8.	Who may need extra iron to prevent deficiency	11
9.	Assessment of iron stores	11
10.	Mechanism of absorption of iron,its regulation, transport and storage	12
11.	Iron toxicity	13
12.	Types of Dietary iron	13
13.	Factors influencing Bioavailability of Heme iron	14
14.	Factors influencing Bioavailability of non Heme iron	15
15.	Effect of simple processing of foods on the Bioavailability of iron	21
16.	Combinations and proportions of foods	24
17.	Increasing intake of foods with more iron bioavailability	25
18.	Conclusion	26
19.	Discussion	28
20.	Reference	29

BIOAVAILABILITY OF DIETARY IRON

Introduction

The nutritional status of a population is one of the factors that significantly affect its health and productivity and consequently the economic development of the community. Among the nutritional deficiencies, iron deficiency is a global nutritional problem, especially in the developing countries. The main explanation for this deficiency is the low bioavailability of dietary iron in the plant based diets that are typically consumed in many developing countries. Iron deficiency reduces mental development in infants and cognitive capacity in school children and decreases immune system capability and working capacity in adults. It also adversely affects pregnancy outcomes, producing, for example, increased maternal morbidity and mortality, premature delivery and low birth weight of infants.

According to Epsy (1977) Bioavailability is the degree to which food nutrients are available for absorption and utilization in the body. Thus bioavailability refers to the amount of a nutrient in a food that the body may ultimately use to perform specific physiological functions.

Iron deficiency is most likely to occur when iron requirements are greatest i.e. during pregnancy, childhood and the reproductive age. In the developing countries, apart from increased requirements the commonest causes of iron deficiency are poor availability of dietary iron and increased iron losses due to parasitic infestations.

In India iron deficiency anaemia is caused primarily by dietary deficiency of iron. The main source of iron from typical Indian diet is contributed by cereals and pulses. Both pulses and grains are fairly good sources of iron (2-10 mg per cent). The prevalence of iron deficiency anaemia therefore in the majority of Indians is paradoxical.

In developed countries iron deficiency is less common but when it occurs it may be due to inadequate dietary iron intake associated with reduced energy

intake or due to the presence of some pathological state interfering with iron absorption or producing increased iron losses.

Iron deficiency anaemia is more common in both urban and rural population of India, inspite of seemingly adequate intake of dietary iron. This may be due to poor absorption, caused by dietary inhibitors like phytates and tannins. Interplay of these inhibitors and enhancers like ascorbic acid determines the availability of iron from the diet.

A factor just as important as the total iron content of the diet is the bioavailability of iron ingested. The absorption of dietary iron is highly variable and depends on what other foods are eaten with the meal, especially on the balance between foods that promote and those that inhibit iron availability.

Prevalence and effects of iron deficiency anaemia in India:

Iron deficiency is recognized as the most prevalent nutritional deficiency worldwide, and children below 3 years of age are considered as one of the highest risk groups i.e. 74% of children in India are with iron deficiency anaemia.

85 per cent of expectant mothers are mild and moderately anaemic and 10 per cent are severely anaemic. Iron deficiency is more common in females (60-70%) than in males (38%). (ICMR,2001)

- Pregnant women belonging to lower socioeconomic group 50-90% during the 3rd trimester of pregnancy are suffering with iron deficiency anaemia
- About 6,00,000 women deaths/year in pregnancy and childbirth are due to nutritional anaemia (Ramakrishna, 1997).
- Prevalence of anaemia is greatest within the country. Lowest prevalence of 33% from Andhra Pradesh. Highest prevalence of 98% in Rajasthan (Seshadri, 1997).

In Kerala 23 per cent women are anaemic and 43.9 per cent children are anaemic (ICMR, 2001).

Distribution of iron in the body

(Helen, 1999)

Type of iron	Percentage
<u>Functional</u>	
Haemoglobin	60-70
Myoglobin	3-4
Tissue iron (enzyme)	5-15
<u>Storage and transport</u>	
Storage iron (liver, spleen and bone marrow)	15-30
Transport iron (transferrin)	0.1
Serum ferritin	<1

Functions of iron in the body:

- Intervenes in the constitution of Haemoglobin and myoglobin.
- Helps combat heavy metal poisoning.
- Maintains immune system
- Acts as a cofactor for several enzymes for proper functioning of cells

Iron plays a vital role in:

- Conversion of β carotene to active form Vitamin A
- Synthesis of purines which forms an integral part in DNA synthesis
- Synthesis of carnitine a vitamin like substance which helps in the transport of fattyacids
- Synthesis of collagen a protein in our body.
- Detoxification of drugs and toxic compounds from liver and intestine
- Synthesis of neurotransmitter like dopanine, serotonin

Effects of iron deficiency

Iron deficiency causes

- Reduced work capacity
- Frequent infection
- Reduced workability and lack of concentration

- Increased distractibility and "pica"
- Shorter attention span and dullness
- Decreased persistence and decreased voluntary activity.

Signs and symptoms of Iron deficiency

Clinical symptoms

- Constant fatigue and lassitude
- Giddiness
- Dimness of vision
- Headache
- Lower resistance to cold
- Insomnia
- Pallor of skin
- Palpitations, rapid pulse rate, Anorexia nervosa, Tingling and pin and needless in the fingers and toes.
- Oedema of the ankles
- Koilonychia
- Glossitis

Evaluation of iron deficiency (WHO, 1968)

Hb norms for adults (in India)

Age/sex group	Hb level (g/dl)
Children 6 months - 5 years	<11
Children 6-14 years	<12
Adult male	<13
Adult females (non pregnant)	<12
Adult females (pregnant)	<11

These levels drop only after iron stores are depleted and deficiency is severe

Iron deficiency and its causes:

Microcytic hypochromic anaemia

If there is an insufficiency of iron for the formulation of haemoglobin, the red blood corpuscles are pale and small and the anaemia is said to be hypochromic and microcytic.

Normocytic normochromic anemia

It is the result of acute blood loss or haemolytic anaemia or bone marrow failure.

Causes of Iron deficiency Anaemia

- 1) **Low dietary intake of iron:** Inadequate intake of iron is secondary to a poor diet like vegetarian diet in which availability of iron from such diets is poor.
- 2) **Failure in utilization:** Failure in utilization is secondary to chronic gastro intestinal disturbances, defective release of iron from iron stores into plasma and defective utilization of iron owing to a chronic inflammation or other chronic disorder.
- 3) **Blood loss:** Blood loss can occur in accidental haemorrhage, in chronic diseases such as TB, ulcers or excessive blood loss due to hookworm infestation.

A heavy infection with hookworm is a common cause of anaemia, where there is a wet cultivation of land. Haemorrhage occur at the site of attachment of the worms to the intestinal nervous membrane. Each worm may ingest adequate quantity of blood per day.

- 4) **Failure to provide for the increased demands:** For infancy and childhood for their rapid growth and for pregnant and lactating mothers where overall 500-600 mg of iron is required during entire pregnancy, which means that a daily requirement of 4-6 mg of absorbed iron is necessary. About 240-400 mg are conserved during the 9 months for the lactation period.
- 5) **Parasitic infestation.** An attack of malarial fever, especially when due to *plasmodium falciparum* is always accompanied by haemolysis and in a severe or prolonged attack, severe anaemia may ensure.
- 6) **Malabsorption of iron:** Inadequate absorption of iron can occur in diarrhea or when there is lack of acid secretion by the stomach or in chronic renal diseases.

when antacid therapy is given. Gastrectomy decreases HCl and transit time through the duodenum.

Iron requirements:

Iron requirements are influenced by the availability of iron present in foods. Iron present in cereals, legumes and green leafy vegetables is available to a lesser extent (due to the presence of phytates and oxalates) than that present in egg, meat and fish (Swaminathan, 1976).

The iron requirements of an adult woman is much higher because of the loss of 2 mg of iron per day in the menstrual blood.

In the case of infants, the iron requirement should not only compensate for basal iron losses, but also provide for an increase in hemoglobin mass and for iron content of body tissues associated with growth.

RECOMMENDED DIETARY INTAKE OF IRON (ICMR, 1989).

Group	Particulars	Iron (mg/d)
Man	-	28
Women	-	30
	Pregnancy	38
	Lactating	30
Infants	0-6 months	-
	6-12 months	-
Children	1-3 years	12
	4-6 years	18
	7-9 years	26
Boys	10-12 years	34
	13-15 years	41
	16-18 years	50
Girls	10-12 years	29
	13-15 years	28
	16-18 years	30

Who may need extra iron to prevent deficiency

- Pregnant women
- Preterm and low birth weight babies
- Toddlers and children
- Teenage girls
- Women of child bearing age
- People with renal failure
- People with gastro intestinal disorders
- Sports persons

Assessment of iron stores

Assessment of iron stores is a more sensitive indicator of IDA.

- 1) **Blood Hb level:** Iron deficiency develops gradually and usually begins with a negative iron balance, when iron intake does not meet the daily used for dietary iron. This negative balance initially depletes the storage form of iron while the blood Hb level, a marker of iron status, remain normal
- 2) **Blood transferrin level:** Transferrin is an iron carrying protein and blood transferring level increases when iron stores are low in the body.
- 3) **Total iron binding capacity (TIBC)** It is normal content of plasma and increases when iron stores are low.
- 4) **Per cent transferrin saturation.** Per cent transferrin saturation decreases when the body iron stores are low. It decreases from normal 30% to 15%

Regulation of iron absorption

- a) **Need for iron.** Iron absorption is tightly regulated, depends on the need. Absorption is increased when new red cells are being rapidly formed in the bone marrow.

Eg: After haemorrhage, iron deficiency anaemia

During pregnancy iron absorption increases to meet the needs of the fetus.

The iron absorption in adolescent girls is 5% as against 3% in boys and girls of other ages.

- b) **Transferrin synthesis:** Iron absorption is mostly regulated through a transport protein- transferrin.

Iron deficiency individuals synthesize more transferrin. Only 20-33 per cent of the iron binding sites of transferrin are normally saturated with iron. Excess iron which is not transferred to transferrin is taken up by the iron storage protein, apoferritin and stored as ferritin in mucosa.

- c) Unbound or free transferrin: Individuals with poor iron status have higher free transferrin levels to pick up more Fe from the gastrointestinal tract. If more iron is needed by the body it is picked up by the unbound or free transferrin.

The ingested iron is solubilized and ionized largely by the acid gastric juice, ascorbic acid, sugars and amino acids which promote absorption, but it is most efficient in the upper portion (Conrad, 1970). Most of the iron absorbed into the blood stream passes rapidly through the mucosal cells in the form of small molecules, that portion exceeding the rapid transport capacity combines with apoferritin form ferritin. Some of the ferritin iron may rather be released for uptake into the blood stream, but most of it seems to remain in the mucosal cells until they are desquamated at the end of their 2-3 day lifespan.

Absorbed iron is transported in plasma, bound to transferrin and deliver this iron to the bone marrow for haemoglobin synthesis, to reticuloendothelial cells for storage. The transferrin content of normal plasma is expressed as total iron binding capacity (TIBC).

MECHANISM OF ABSORPTION OF IRON, ITS REGULATION, TRANSPORT AND STORAGE

Iron absorption is defined as the percentage of ingested iron that after the digestion of food, has passed the mucosal cell and is taken up by the blood. During each stage of absorption, iron interacts with other components of ingested food. These interactions influence absorption, bioavailability and utilization of iron positively or negatively (Dokkum, 1992).

Iron absorption takes place mainly in the Duodenum and upper jejunum of intestinal mucosal cells (Muir, and Hopter, 1985). Iron enters the stomach from the oesophagus. Iron is oxidized to the Fe^{3+} state no matter its original form which

takes orally. Gastric acidity as well as solubilising agents such as ascorbate prevent precipitation of the normally insoluble Fe^{3+} . Intestinal mucosal cells in the duodenum and upper jejunum absorb the iron. The iron is coupled to transferrin (TF) in the circulation which delivers it to the cells of the body.

Iron toxicity/Iron overload

Iron toxicity is a condition in which excess iron is found in the blood and stored in organs such as liver and heart.

Hemosiderosis or Siderosis

This deficiency occurs when there is abnormal destructions of the red blood cells as in hemolytic anaemia. An excessive intake of iron daily produces typical symptoms of cirrhosis of the liver as the iron is stored in it in large amounts.

Hemochromatosis

An inborn error of metabolism in which there is a failure to control iron absorption from the small intestine. It continues in an unregulated fashion causing iron overload.

Characteristic Features

- Enlarged and cirrhotic liver
- Pancreatic diabetes
- Slate gray discolouration of the skin
- Hypogonadism

Types of dietary iron

There are two distant types of dietary iron heme and non heme iron. Heme iron is a constituent of haemoglobin and myoglobin and therefore is present in meat, fish and poultry, as well as in blood product. Hence iron accounts for relatively small fraction of total iron usually less than 1-2 mg of iron per day. The second type of dietary iron, non heme iron, is the most important source. It is found to varying degrees in all foods of plant origin. The absorption of iron from a diet is thus determined by the amounts of heme and non-heme iron present in the diet (Hall berg and Rossander, 1991).

Sources of dietary iron.**Sources of dietary iron:**

Chemical form and types of iron	Source
Heme iron	Meat, fish, poultry and blood products Bioavailability is high - 20 to 30%
Non heme iron	Mainly found in cereals, tubers, vegetables and pulses Bioavailability determined by the presence of enhancing and inhibitory factors concerned in the same meal
Contamination iron	Soil, dust, water, iron pots etc. Potential bioavailability usually low. May be present in large quantities, in which case its contributions to total iron intake is not insignificant

Factors influencing bioavailability of dietary iron :

Bioavailability is the percentage of ingested iron that becomes available for metabolic action. Bioavailability includes iron absorption, transport of iron to relevant body tissues, and conversion into its physiologically active forms (Dokkum, 1992).

Iron absorption refers to the amount of dietary iron that the body obtains and uses from food. Healthy individuals absorb about 10-15% of dietary iron but individuals' absorption is influenced by several factors.

Factors influencing bioavailability of heme iron**Individual factors**

Heme iron absorption is much less influenced by the iron status of the subject. Partial gastrectomy and pathological conditions that affect the small bowel mucosa, such as coeliac diseases, do not significantly affect the absorption of heme iron.

b) Dietary factors

Meat greatly facilitates the absorption both non heme and heme iron from the diet. Most of the iron in the liver is heme iron and remaining 30 percentage is mainly ferritin and hemosiderin. The bioavailability of heme iron in meals containing meat is about 25 per cent and the bio-availability of heme iron given without meat or liver has a maximum absorption of about 10 per cent decreasing with increasing dose to a five per cent.

Factors influencing the bioavailability of non heme iron

a) Individual factors

(I) Anaemia

Nutritional anaemia may be defined as the condition that results from the inability of the erythropoetic tissue to maintain a normal haemoglobin concentration leading to reduction in the total circulating haemoglobin.

- (i) Iron deficiency
- (ii) Hemolytic
- (iii) Acute blood loss
- (iv) Sideroblastic refractory

(II) Pregnancy

The bioavailability of iron increases during pregnancy and is roughly parallel to the increased iron requirements (Hallberg, 1987)

(III) Disease states

In idiopathic hemodermatosis, the absorption of food iron is markedly increased in relation to the size of iron stores (Benzweda *et al.*, 1978)

Other factors which influence iron absorption are hypoxia, erythropoietin administration, cobalt administration, liver regeneration, pyridoxin deficiency etc.

b) Dietary factors

Enhancers which include organic acids like ascorbic acid, succinic acid, and citric acid, proteins, sugars & fat.

Inhibitors are phytates, tannates, fibre, calcium, soyabean, oxalates and phosphates.

1) Ascorbic acid

Ascorbic acid is a powerful promoter of non-heme iron absorption. About 75 mg ascorbic acid increased non heme iron absorption three to four fold (Cook and Monsen, 1977). When ascorbic acid was added to a meal, the increase in iron absorption was proportional to the dose of supplementation (Cook and Monsen, Lynch and Cook, 1980).

According to Deehr *et al.*, (1990) ascorbic acid present in orange juice consistently increased non heme iron absorption. Higher percentage of dietary ascorbic acid content in high socio-economic group when compared to the low socio-economic group might be one of the several factors which caused the higher availability of dietary iron and increased blood iron and haemoglobin in high socio-economic group than the low socio-economic group (Annapurani and Murthy, 1984).

Ascorbic acid is the best enhancer of non heme iron absorption. The mechanism by which Vitamin C promotes iron availability by reducing ferric iron to its ferrous oxidation state and secondly ferric iron may form a ferric ascorbate complex at the low pH prevailing in the stomach. This iron chelate remains stable and soluble at the higher pH in the duodenum, thus making iron available for absorption. This complexing, however, must be initiated at a low pH (Lynch and Cook, 1980, Clydesdale, 1981).

A study conducted by Hunt *et al.* (1990) showed that in iron depleted women consuming a diet with predicted poor iron availability, ascorbic acid supplementation enhanced body iron retention for 5.5 weeks.

Annapurani and Murthy (1984) concluded that the rise in serum iron and haemoglobin level after supplementing spouted green gram in the diet is due to an increase in the ascorbic acid content.

2) Succinic acid

Succinic acid which increases the absorption of iron from pharmaceutical doses of iron had about the same 35 per cent absorption promoting effect on dietary non heme iron (Rasmussen, 1974).

3) Citric acid

Citric acid also enhances iron absorption *in vivo* and *in vitro* (Gillooly *et al.*, 1983). Both fructose and citric acid have been associated with enhanced iron availability for absorption through the formation of soluble chelates (Bates *et al.*, 1972; Hazell and Johnson, 1987).

4) Proteins

The influence of food protein products on non-heme iron bioavailability is known to vary according to the protein product (Cook and Monsen, 1980). Various aminoacids are capable of dissolving metal phosphates precipitates and hence making iron more available for absorption (Dokkum, 1992).

Several free amino acids enhances iron absorption. Histidine, valine and lysine have been shown to have an enhancing effect on iron absorption when given simultaneously with iron. Even sulphur containing amino acids may also have an effect in facilitating iron absorption. Amino acid supplementations to dietary protein enhanced the absorption and utilization of iron in rat.

5) Sugars

Some sugars, example fructose and lactose form soluble chelates of iron in the intestinal lumen that favour iron absorption whereas glucose and galactose has no effect on iron absorption (Pannell *et al.*, 1979).

Iron absorption is considered to be modified by the sugars. Nature of carbohydrates in enhancing the absorption of iron is as follows: lactose > sucrose > glucose > starch. Starch diet markedly inhibited absorption when compared to the diet containing glucose or disaccharides, especially lactose which appeared to promote more efficient iron utilization.

6) Fat

Amount and type of dietary fat also modify the absorption of inorganic iron. Absorption of iron was greater in diets containing coconut oil as a source of fat than in those containing corn oil. Besides this it was also found that diets higher in fat favoured iron absorption (Amine and Hegsted, 1975).

Inhibitors of iron absorption

a) Individual factors

In gastric achlorhydria the absorption of dietary non-heme iron is reduced in relation to the absorption from a ferrous iron salt. After partial gastrectomy a decrease in the bioavailability of non-heme iron is often observed. The magnitude of the decrease depends on the type of gastric operation performed.

b) Dietary factors

Many compounds in foods are reported to inhibit the absorption of iron, among them are phytates, oxalates, phosphates, polyphenols and soy products (NIN, 1990). Iron from vegetable origin was much more 'surrounded' by inhibitors, such as phytates, fibre, oxalate and phosphates, than iron from animal origin (Dokkum, 1992). According to Hallberg and Rossander (1991) the important factors that decreased the bioavailability were inositol phosphate (eg. phytate), iron binding phenolic compounds and calcium. Thus iron absorption is adversely affected by the presence of the following dietary factors.

1) Tannates

Derman *et al.*, 1987 reported that tea markedly reduced the absorption of non-heme iron from foods. This effect has been ascribed to the formation of iron tannate complexes. It has also been suggested that tannins may be partly responsible for the low bioavailability of iron in many vegetable foods.

Many popular beverages such as tea and coffee also constitute an important source of iron binding phenolic group such as tannin compounds. These compounds were considered to be responsible for the reduced bioavailability of non heme iron from foods (Brown *et al.*, 1990). Compounds with a polyphenolic structure are considered responsible for the reduced availability of iron because of the formation of insoluble complex (Dokkum, 1992).

Separate the tea drinking habit from the meal time. One or two hours later, the tea will not inhibit the absorption, since most of the food would have left the stomach. Tamarind, turmeric and chilies were rich sources of tannins.

2) Phytates

The diets of Indian people are based on cereals and contain excessive phosphorus and phytates and low calcium. It has been postulated that phytate interference with the absorption by complexing with iron as it passes through the intestinal tract and it not available for absorption.

According to Hallberg and Rossander (1991) the important factors that decreased the bioavailability were inositol phosphates (eg. Phytates); iron binding phenolic compounds and calcium. The potent inhibitors of non heme iron absorption widely distributed in vegetable foods were phytates and polyphenols. Phytates in whole grain cereals, legumes, nuts and seeds could bind non heme iron and greatly reduce its absorption. Phytic acid prevent as a constituent in many cereals reduce iron absorption by forming insoluble complex of ferric phytate which was made soluble during germination (Annapurani and Murthy, 1984).

The solubility of non heme iron in the small intestine is a major factor in determining its absorption. Polyphenols and phytates in plant foods are known to bind with non heme iron and thus inhibit its absorption (Craig, 1994).

3) Fibre Fibre was responsible for the low bioavailability observed from cereals. Binding of divalent metal ions such as iron (Fe), calcium (Ca) and Zinc (Zn) by dietary fibre was been demonstrated by several investigators. As a result, decreased intestinal mineral absorption is one of the possible negative effects of dietary fibre (Rao and Ramulu, 1998). Study conducted by Jainita (1998) showed that the crude fibre reduced from 4.08 g/100 g to 3.97 g/100 g, 3.85 g/100 g and 3.90 g/100 g on 24 hours, 48 hours and 72 hours of germination respectively. On dehulling too the fibre content reduced from 4.20 g/100 g to 1.35 g/100 g. Thus the low bioavailability of iron in ragi inspite of its high iron content may be attributed to its high fibre and tannin content.

4) Phosphates. The addition of egg white to favour ascorbate reduced iron absorption. The vitellin fraction of yolk, high in phosphoprotein, was believed to amount for this absorption. Although egg contain approximately 1 mg of iron, the iron is poorly available to humans and the egg yolk further inhibits absorption of other dietary iron (Bayness and Bothwell, 1990). Yolk phosphates of egg binds

ionic iron and reduces its solubility thus decreasing its availability to mucosal cells (Halkett *et al.*, 1980).

5) Oxalates: Oxalates decreases iron absorption. Studies on meal served with and without extra oxalic acid (100 mg) have failed to show such an effect. Green leafy vegetables and other vegetables contain oxalic and which inhibits iron absorption. Green leafy vegetables has low total iron content but higher ionisable iron (35%) when compared to pulses (NIN, 1994).

6) Calcium: Supplemented CaCO_3 has been shown to inhibit iron absorption. In humans when it was consumed together with food, CaCO_3 supplements depressed iron availability. Both the cation and the anion in CaCO_3 contributed to the iron absorption -depressing action of this salt (Prather and Miller, 1992).

A study by Kochanowski and Mc Mahan (1990) demonstrated that Calcium inhibited iron absorption when two minerals were present together but when reported by one hour, there was no inhibition. This suggested that calcium has to be present in the same meals as iron to interfere with its absorption. About 30-50 per cent more iron was absorbed when no milk or cheese was served with lunch or dinner. A reasonable separation of calcium and iron intake would improve iron nutrition (Gleerup *et al.*, 1995).

A study by Deehr *et al.* (1990) had concluded that 500 mg calcium as milk had an antagonistic effect on iron absorption.

7) Zinc and Copper Zinc and copper may bind to thioneins forming the respective metallothioneins but copper binds more strongly than zinc. Hence the transport of copper across the cell is impaired and less copper enters the circulation. As may be the case with iron bound to ferritin in the mucosal wall, the copper - thionein complex is released into the lumen by desquamation of the cell. Because copper is involved in iron metabolism as ferroxides, a relatively high intake of zinc may therefore induce anaemia (Dokkum, 1992)

8) Soy proteins Soy proteins or soy flour is known to reduce the absorptions of non-heme iron in humans.

Macfarlane *et al.* (1990) showed that when compared with a soy flour meal containing an equal amount of protein, iron absorption was found to be

significantly improved. This improvement could be explained with reference to a change in the protein composition of the product due to fermentation.

Effect of simple processing of foods on the bioavailability of iron.

1) Milling

Milling of cereals greatly affects their iron bioavailability. Iron in milled polished rice, for instance is about 4 times better absorbed than is iron in unmilled rice. Study conducted by Jainita (1998) in KAU reported that in processing rice the total iron was found to be maximum in the case of parboiled and hand pounded rice while the absolute available iron was more for parboiled milled rice due to the reduction in the fibre content as a result of milling.

2) Dehulling

Use of pulse after decortication to dhal is a most common household practice. The outer seed coat of the pulse grains have the polyphenolic compounds such as tannins which interferes in the absorption of iron. Study conducted by Jainita (1998) revealed that on dehulling pulses both total and absolute available iron was found to increase except in the case of soyabean. This can be attributed to the fact that decortication of seeds reduced tannin to a low level with a significant increase in the ionisable iron. Dehulling of pulses substantially reduced the fibre simultaneously increasing the total iron content

Studies conducted at NIN (1994) revealed that the total iron content in seven varieties of whole pulses ranged between 5.7-6.4 mg/100 g but the per cent ionisable iron varied widely from 3.4 to 35.6. The per cent ionisable iron in split pulses was found to be significantly higher. There was no difference in the value of phytate between whole and split pulses but the tannin content was found to be low in split pulses

3) Germination

During germination, several enzyme system become active and bring about profound changes in the nutritive value of pulses. Antinutritional factors such as phytates and tannins which conversely affect the bioavailability of divalent ions are broken down on germination. Germinated seeds were analysed before and after decortication.

During germination the tannin content decreased by 20-30 percentage while ionisable iron nearly doubled. Decorated seed samples which had low tannin and high ionisable iron did not show any improvement at different stages of germination. The phytate content of the seeds decreased and ascorbic acid content increased during germination. A reduction in tannin content of the whole seed during germination is responsible for the observed improvement in ionisable iron (Rao and Prabhavati, 1982).

Study conducted by Jainita (1998) indicated that when pulses were germinated, the total and absolute available iron increased with increase in time of germination. This may be due to the breakdown of antinutritional factors such as tannin and phytates during germination. The effect of germination of ragi on tannin and ionisable iron revealed that the tannins reduced with increasing hours of germination. On germinating ragi the total and absolute available iron were found to increase with a simultaneous reduction in the fibre content upto 48 hours of germination. Pulses do not contain ascorbic acid other than when they are germinated thus, with increase in germination time of ascorbic acid, total and absolute available iron were seen to increase

4) Roasting and puffing

Roasting and puffing results in imparting a describable flavour and taste. It also destroys the antinutrients which are thermo labile which inturn favour absorption. Roasting meat products resulted in high levels of non heme iron after cooking compared with before cooking (Schricker and Miller, 1983)

When the effect of different cooking methods were analysed, the total and the absolute available iron seen to increase on roasting black gram and Bengal gram the absolute available iron increased ten to fifteen fold. A further increase was observed in both total and absolute available iron when pulses were roasted after dehulling (Jainita, 1998)

5) Fermentation

Research evidence indicate that the process of fermentation modifies the nutritional value of food by increasing the bio-availability of nutrients

Study conducted by Reddy *et al.* (2000) showed that there was a fall in phytate phosphorus content and an increase in tannin content were noticed with a rise in the period of fermentation. Fermenting foods beyond the period of six hours enhanced the content of ionisable iron and also the bioavailability of iron significantly.

A reduction in the content of antinutrients was noticed in idli and ~~khaman~~ dhokla as compared to that in batters of idli and khaman dhokla. On the other hand, the ionizable iron content as well as bio-availability of iron did not differ significantly between idli and idli batter. In khaman dhokla the ionizable iron content was significantly more than that in dhokla batter but the bioavailability of iron remained almost the same.

Study conducted at KAU by Jainita (1998) showed that there was a slight increase in the absolute available iron of fermented and unfermented idli batter which further increased. In the case of prepared idli. Thus iron became more available on fermentation and cooking by steaming.

(6) Cooking

Braising meat resulted in significantly higher non heme iron level on the surface portion compared with before cooking levels and with the centre portion after cooking. The microwave methods did not significantly affect the distribution of non heme iron to total iron before and after cooking. The braising and roasting methods cooked the meat to a higher interval temperature and resulted in higher non heme iron levels than the microwave method (Schricker and Miller, 1983). Heating foods and storing cooked foods easily destroyed ascorbic acid and therefore significantly decreased iron availability for absorption. Prolong storage of canned products increased the iron content of foods. Reduced heme iron absorption has been observed after over cooking of meat causing denaturation of the heme (Martinez *et al.*, 1986).

The total iron and the absolute available iron was maximum for pressure cooked rice rather than rice boiled by excess water method. This increase in the total and absolute available iron may be due to the loss of inhibitory factors during cooking under pressure. When pulses were boiled after soaking the total

and absolute available iron increased due to the leaching out of appreciable quantities of tannin thus enhancing the availability of iron after cooking. A further increase in both total and absolute available iron was observed on pressure cooking of soaked pulse (Jainita, 1998).

(7) Cookware used

Addition of iron to foods due to preparation in iron cookware has often been cited as being significant. Although the possible role of iron cookware in iron nutrition has been debated, some studies have shown that intake of food exposed to iron cookware can produce a significant raise in haemoglobin values in humans. However, no biological studies for such nutritional iron have been found in the literature (Rosenoff and Kennedy, 1982).

Study conducted by Jainita (1998) on the effect of different cooking vessels on the total and absolute available iron, observed by different cooking and processing methods revealed that the total iron was maximum for cereals and pulses cooked in iron vessels mainly due to the contamination of iron from the iron cookware. The absolute available iron was maximum for cereals and pulses cooked in inert vessels like glass, mud and tufflon coated vessels and it was least in the case of aluminium vessels which react with food constituent forming complex compounds which makes the iron unavailable

Combinations and proportions of foods

The food combinations were formulated on the basis of using foods that are normally consumed familiar, locally available and low-cost, containing enhancing factors and limiting inhibitors to the extent possible and providing an overall balanced diet to provide all the major nutrients required by the body (NIN, 1992-93)

- Tea with milk, lemon tea or herbal tea has been included between meals (and not with them) for better absorption from the meals
- Restrict beverages along with the main meals. Milk is taken at breakfast, in the evening or at bed time and not with the main meals that contribute most of the daily iron intake. Milk intake may be increased to 400 ml per day provided it is distributed as suggested

- Include Jaggery instead of sugar as it contains iron whereas sugar contains only a trace.
- Early morning tea may be deleted and replaced with mid-morning or mid-afternoon tea.
- Meals may be cooked as one dish, eg. missiroti (chapatti) comprising wheat or millet flour + pulse flour (chickpea) + GLV or gingelly seeds; or rice khichdi consisting of rice + green gram or chickpea split pulse + greens to improve nutritive value by supplementary action and to reduce cooking time.
- Eat vegetables like cabbage, radish leaves and capsicum after food-safety aspects have been observed, should preferably be eaten raw as salads to avoid loss of their vitamin C content.

Increasing intake of foods with more iron bioavailability

The overall intake of iron from iron rich foods needs to be increased to obtain the optimum level of RDA of iron in Indian population groups. This increase should be coupled with efforts to combine appropriate foods in the diet to enhance the bioavailability of iron and reduce inhibitory factors.

- Processing of cereals and pulses like milling, dehulling, germination, fermentation etc. enhances bioavailability.
- Cereals and millets, pulses and legumes, GLV, nuts and oilseed are good sources of iron. Even without the haem iron found in fish or poultry, vegetarians are not at greater risk from iron deficiency than non-vegetarians (Miller, 1999).
- Plant foods can supply all the haematinic nutrients in adequate amounts with the exception of vitamin B₁₂.
- Encouraging production, processing, marketing and consumption improves dietary consumption of iron.
- Vitamin C rich foods must be consumed at the same meal that contributes the major part of daily dietary iron (Cook and Monsen, 1977).
- Household processes such as germinations, matting of grains/pulses and fermentation should be used to overcome phytates (Choudhary and Vir, 1994) and enhance the ascorbic acid and B vitamins.

- The presence of carotene in rice, wheat and corn based diets improved iron absorption from one to more than three fold suggesting that both ascorbic acid and carotene (Garcia-Casal *et al.*, 1998) prevented the inhibitory effect of phytates on iron absorption.
- Separate tea drinking habit from the meal time which contains inhibitors like tannates reduces the bioavailability.
- Avoid taking calcium and iron rich foods at the same time (Hallberg *et al.*, 1991; Hallberg, 1998) 300-600 mg of calcium inhibited iron absorption by 60 per cent, which is the maximum inhibition of iron.
- Eggs should be eaten along with a fruit. They are rich in iron content, but its bioavailability is poor. When two eggs were consumed per day there was a significant reduction in iron absorption from Indian meals (Kaur, 1981).
- Inclusion in our daily about 50 g of GLV, children who consumed GLV once a week or more frequently had higher iron levels than non-consumers (Seshadri, 1997).
- Daily intake of fruits rich in vitamin C with two major meals for eg guava results in a significant increase in iron (Seshadri *et al.*, 1985).

Conclusion

Enhanced production and consumption of animal foods would be a solution to the problem of poor availability of dietary iron as heme iron are readily absorbed and they also enhance non heme iron availability. However, the present limitations in the production and availability of foods of animal origin, the low purchasing power of the majority of the people and cultural constraints on intake of animal foods are formidable barriers. It is probable, therefore, that for a long time to overcome the need for iron will be met only through nonheme iron source.

It is not practical to substitute part of the plant foods by animal foods (Heme Iron) due to their higher cost and cultural constraints therefore alternative approaches of increasing the total bioavailable iron intake are necessary as strategies for combating iron deficiency anaemia (Kakade and Agti, 1997)

From the results of the study it was concluded that substitution or supplementation of locally available, low cost foods high in bioavailable iron to

rice based diets would enhance the absolute available iron of our diet and such combinations require to be encourage

DISCUSSION

1. Which are the green leafy vegetables rich in iron?

(A) Chekkurmanis, radish leaves, amaranthus, kuppameni.

2. Why all animal foods are not rich source of heme iron though they contain heme iron?

(A) meat, fish, poultry are good source of heme iron, it is found that 1g of meat has the same enhancing effect as does 1mg of ascorbic acid. The protein must not only be animal protein but must be contained within animal tissue. Hence animal protein in milk, cheese and eggs does not have the same effect.

3. What is pica?

(A) It is the habit of eating inedible things like chalk, clay etc. As I mentioned earlier, certain neurological changes leading to behavioural abnormalities are common in anaemia. Pica is one of them.

4. How can you prevent iron toxicity in a condition like hemochromatosis?

(A) It is treated by reducing the load of iron in the body. This is effectively accomplished by periodic blood withdrawal. Half a litre of blood contains approximately 200-250 mg of iron.

5. What is the difference between soy meal and soy sauce?

(A) Soy sauce is a fermented soy product produced by inoculating the soybeans with various species of bacteria, fungi and moulds whereas soy meal is obtained by grinding the cake or chips that remain after removing most of the oil from the soybeans.

6. How soy bean inhibits iron absorption?

(A) The Bioavailability of iron from soy is influenced by the form of soy product, and depend on whether fibre and phytic acid is present. Soy bean is deficient in methionine which increases iron absorption.

7. Are dates good source of iron

(A) Yes all dried fruits are rich source of iron as they contain sugars.

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ABSTRACT

Bioavailability is the degree to which food nutrients are available for absorption and utilization in the body. Thus bioavailability refers to the amount of a nutrient in a food that the body may ultimately use to perform specific physiological functions (Epsy, 1977).

There are two distinct types of dietary iron, heme and non-heme iron. The bioavailability of iron from a diet is thus determined by the amounts of heme and non-heme iron present in the diet (Hallberg and Rossander, 1991).

The heme iron is more available to the body. It is not affected by any dietary factors

Bioavailability of non heme iron is depended on various enhancers and inhibitors in the diet. The dietary factors affecting the bioavailability of non-heme iron are ascorbic acid, succinic acid, citric acid, protein, sugar and fat. Ascorbic acid is the best enhancer of non-heme iron absorption (Clydesdale, 1981). Various amino acids are capable of dissolving metal phytate precipitates and hence making iron more available for absorption (Dokkum, 1992)

Many compounds in foods are reported to inhibit the absorption of non-heme iron, among them are phytates, oxalates, phosphates, tannates, fibre, calcium and soyproteins. Phenolic compounds were considered to be responsible for reduced bioavailability of non-heme iron from foods (Brown *et al.*, 1990)

Simple processing methods such as nulling, dehulling, germination, roasting, fermentation and cooking increased the total and available iron in foods (Jainita, 1998). Studies conducted by Reddy *et al.* (2000) showed that there was a fall in phytate and phosphorus content with a rise in the period of fermentation and thereby an increase in the availability of iron from fermented foods

It is not practical to substitute part of the plant foods by animal foods (heme iron) due to their high cost and cultural constraints. Substitution or supplementation of locally available low cost foods high in bioavailable iron to rice based diets would enhance the absolute available iron of our diet.

NUTRITION-PROBLEMS AND PROGRAMMES IN INDIA

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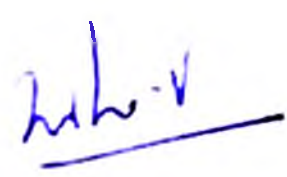
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CONTENTS

SL.No.	Title	Page No.
1.	Introduction	1
2.	Major deficiency diseases in India	2
3.	Protein Energy Malnutrition	3
4.	Nutritional Anaemia	5
5.	Vitamin A Deficiency	6
6.	Iodine Deficiency Disorder	7
7.	B Complex Deficiency	10
8.	Causes and consequences of malnutrition	11
9.	Effect of malnutrition	12
10.	Prevention of malnutrition in India	13
11.	Nutrition programmes in India	14
12.	Food supply and supplementary feeding programmes	15
13.	Vitamin and mineral supplementation programmes	18
14.	Integrated Child Development Services	20
15.	National Diarrhoeal Control Programme	22
16.	National Vector Borne Disease Control Programme	22
17.	Combating vitamin A deficiency and anaemia	23
18.	Conclusion	24
19.	Discussion	25
20.	References	i-ii
21.	Abstract	

NUTRITION- PROBLEMS AND PROGRAMMES IN INDIA

Introduction

In the ultimate analysis, it is the quality of the human resources of a country that determines its strength of greatness; and a major determinant of that quality is the health/nutritional status of its people. Nutrition is a major factor in bringing out the maximum potentiality that one is endowed with both physically and mentally. Good nutrition depends on an adequate supply and this in turn on a sound agricultural policy and a good system of food distribution (Sreelakshmi, 2002).

Three major factors could have militated against any dramatic improvement in the nutritional status of India's population during the last two decades. First, there has been a rapid increase in population, which had nullified the beneficial effects of substantial increase in food grain production. Second, social and economic inequity and 'distributive injustice', which lay the root of the problem of undernutrition. Third, national efforts at ensuring better child survival have not, thus far, been matched by equally vigorous efforts at ensuring better health and nutrition among the survivors.

Despite these limitations, there are heartening indications that, at long last India may be at the turning point with respect to nutrition. The nutritional status of poor children has not deteriorated and indeed has shown some improvement. So there has been no spectacular decline in the overall percentage of people below the poverty line, there has apparently been a significant fall in the proportion of the 'poorest of the poor' - to whose ranks most cases of severe undernutrition generally belong. The percentage of children suffering from 'severe' under nutrition has significantly declined in recent years (Gopalan, 1993).

Malnutrition is an ecological problem that does not occur alone. Its consorts are, poverty, disturbed family structure, ignorance and despair. The term malnutrition includes both undernutrition as well as overnutrition and in developing countries, under nutrition is the major public health problem. The main victims are children and women in reproductive age group (Vazir, 2003).

Diseases like cardiac beriberi, peripheral neuritis, burning feet syndrome and pellagra, which used to be the major public health problems in the 1950s and early

1960s, have disappeared. Kwashiorkor of the classical kind has steeply declined. Keratomalacia, a major cause of nutritional blindness in children till the 1960s has now almost disappeared. The decline of these diseases did not come about because of specific 'intervention programmes' (Reddy *et al.*, 1993).

Some facts about malnutrition and micronutrient deficiencies in India

According to the Department of women and child Development, Ministry of Human Resource Development, 1999;

- Nearly one-third of the world's children suffering from malnutrition are in India.
- Incidence of micronutrient deficiencies, nutritional anaemia vitamin A and iodine deficiencies are still very high
- Rate of malnutrition is falling much too slowly at only one per cent per year.
- More than half of pre-school children are stunted (56.5%) and nearly a similar proportion (49.2%) are under weight (DNP Survey, 1995-96)
- One in every six children is excessively thin (wasted)
- Nearly 16 per cent infants less than 6 months and about 43 per cent infants between six to eleven months are malnourished
- About 30 per cent babies are low birth weight babies
- Nutritional anaemia affects about 50 per cent of young children, adolescent girls and women in the reproductive age group
- More than 10 per cent of population, in 235 districts of India is affected with goitre- an iodine deficiency disorder (Joshi, 2002)

Major Deficiency Diseases in India

The major types of deficiency diseases are

1. Protein Energy Malnutrition (PEM)
2. Nutritional Anaemia
3. Vitamin A Deficiency (VAD)
4. Iodine Deficiency Disorders (IDD)
5. B complex Deficiency

(UNICEF, 1992)

1. Protein Energy Malnutrition (PEM)

PEM is a condition where there is a deficiency of protein and calories in the diet. Its clinical stages ranging from kwashiorkor to mild symptoms like mild growth retardation.

Prevalence

Kwashiorkor	-	0.04%
Marasmus	-	0.07%

(ICMR, 2001)

Aetiology

Diet A diet predominantly deficient in protein leads to kwashiorkor while a diet deficient in calories results in marasmus

Role of free radicals and aflatoxins: In the malnourished children, deficiency of the nutrients such as vitamins A, C and E in the presence of infection or aflatoxin may result in the accumulation of toxic free oxygen radicals, and these may damage liver cells giving rise to kwashiorkor

Infection: Repeated attacks of diarrhoea were shown to be responsible for poor growth of children. Measles is the other common infectious disease that occurs during childhood. Malabsorption of nutrients and metabolic losses during infection can also aggravate malnutrition

Socio economic factors: PEM is basically a problem of poverty, resulting from inadequate diets, poor environment and high incidence of infections. Faulty feeding habits arising from ignorance and prejudices, superstitions and taboos concerning food are powerful social factors that influence nutritional status.

Some important socio-economic factors are

Inadequate breast-feeding

Unhygienic living conditions

Environmental contaminations

Maternal malnutrition

Infectious diseases

↑
Intestinal parasites

Family size

Clinical features

Kwashiorkor

Marasmus

Marasmic Kwashiorkor

Nutritional Dwarfing

Kwashiorkor: The three essential features of kwashiorkor are growth failure, oedema and mental changes.

Growth failure may be masked by oedema.

Oedema: Oedema appears first on the feet and legs and later spreads to the whole body. The face looks puffy with sagging cheeks and swollen eyelids.

Mental changes like apathy and irritability are common features.

Anorexia is common in children making it difficult to feed the child.

Diarrhoea is common in children. It may be due to defective digestion and absorption or because of secondary infection.

Skin and hair changes: The texture and colour of hair changes and it becomes thin, dry and can be pulled out easily without causing pain. Changes in colour include brownish or reddish discolouration. Scaly pigmentation of the skin is common, and in severe cases the epithelium peels off leaving behind depigmented patches with oozing out of fluids, which is described as 'crazy pavement dermatosis'.

Marasmus

It is due to the consumption of diets grossly deficient in both protein and calories. The child looks appallingly thin with shrivelled body, wrinkled skin and bony prominences. Skin and hair changes are infrequent. Oedema and fatty infiltration of liver, the prominent signs of kwashiorkor are absent in marasmus.

Marasmic Kwashiorkor

This is a combination in varying degrees of the features of the two conditions marasmus and kwashiorkor. The malnourished children exhibit the features of marasmus as well as kwashiorkor. They have varying degrees of muscle wasting.

oedema, as well as skin and hair changes. This is due to the varying nature of dietary deficiency and social factors responsible for the disease and absence of infections.

Nutritional Dwarfing

Some children adapt to prolonged insufficiency of food energy and protein by a marked retardation of growth, weight, height etc. are both reduced resembling children a year or more younger. It has found that malnutrition during preschool years results in stunted growth, which affected the later development of the children (Anand, 1994).

2. Nutritional Anaemia

Anaemia is defined as the reduction in the haemoglobin level in circulation because of inadequate supply of one or more nutrients (Raman and Sarma, 2003). Nutritional anaemia is caused by the lack of any dietary factor that is involved in haemoglobin synthesis or by poor absorption of this dietary essential. Some anaemias are caused by lack of either dietary iron or high quality protein, by lack of pyridoxine (vitamin B₆) which catalyses the synthesis of the haem portion of the haemoglobin molecule, by a lack of vitamin C which influences the rate of iron absorption in to the tissues, or by a lack of vitamin E which affects the stability of the red blood cell membrane. Children, adolescent girls and pregnant women are the most vulnerable groups

Prevalence

In India 20-70% of the population are suffering from various degrees of anaemia. Prevalence of anaemia in children 74%, adolescent girls 60-70%, and in of expectant mothers 85% are mild and moderately anaemic and 10% are severely anaemic. Anaemia is more common in females than in males, which is 38% (ICMR, 2001). A study George *et al* (2000) found that the prevalence of anaemia in preschool children in Kerala is 11.4%. The problem of anaemia is worldwide. Soerkirmn and Jalal (1991) reported that nearly 200 millions of preschool children suffered from anaemia and they had learning disabilities which resulted in sub optimal, scholastic performance

Aetiology

- Inadequate intake and reduced availability

- Inadequate utilization of iron because of gastro intestinal disturbances and chronic inflammations.
- Blood losses due to accidental hemorrhages, tuberculosis, ulcers, or intestinal disorders, hookworm infestation, etc.
- Inadequate absorption can occur in diarrhoea

Symptoms

- Functional disturbances: Impaired cell mediated immunity, reduced resistance to infection, increased morbidity and mortality and diminished work performance
- In more severe IDA Defects arises in the structure and function of the epithelial tissue - tongue, nails, mouth and stomach, the skin may appear pale, inside of the lower eyelid may be light pink.
- Koilonychia: Fingernails can become thin and flat (spoon shaped nails)
- Mouth: Atrophy of the lingual papillae, burning redness and in severe cases completely smooth waxy and glistening appearance of the tongue called glossitis, angular stomatitis, dysphasia and gastritis
- General symptoms: Lassitude, fatigue, breathlessness on exertion, Palpitations, dizziness, tinnitus, headache, dimness of vision, insomnia, etc

3. Vitamin A Deficiency (VAD)

Vitamin 'A' deficiency is more prevalently seen in pre-school children. It affects many tissues in the body; the most dramatic changes are seen in the eyes resulting in tragic consequences of total loss of vision in early life (Vijayaraghavan, 1989).

Prevalence

Children <6 years

Bitot's spots - 0.70%

Night blindness - 0.71%

Pregnant women

Night blindness - 2.76%

(ICMR, 2001)

Aetiology

- Inadequate dietary intake of vitamin A or its precursor (β -carotene)
- Inadequate dietary intake of vitamin A -by mother during pregnancy.
- Inadequate intake of vitamin A rich food during weaning period.
- High cost of animal foods like egg, milk and liver - lead to vitamin deficiency.
- Infections like measles, diarrhoea, respiratory tract infections etc.
- Lack of awareness - diarrhoea control, immunization, vitamin A supplementation and other health services.

Symptoms

Night blindness Night blindness is an early symptom of vitamin A deficiency. In VAD the individual cannot see objects in dim light. In vitamin A deficiency the formation of rhodopsin is impaired giving rise to night blindness.

Conjunctival xerosis The conjunctiva is dry, thickened, wrinkled and pigmented due to keratinisation of the epithelial cells. The pigmentation gives the conjunctiva a smoky appearance.

Bitot's spots It is more an extension of xerotic process. Greyish glistening white plaques formed of desquamated thickened conjunctival epithelium, usually triangular in shape and firmly adhering to the conjunctiva.

Corneal xerosis When dryness spreads to cornea, there is a dull hazy lusterless appearance. This is due to the keratinisation of the epithelial tissue covering the cornea.

Keratomalacia Xerosis of the conjunctiva and cornea may lead to keratomalacia. In this condition softening and dissolution of the cornea occurs. The perforation of the cornea leads to prolepses of the iris, extrusion of the lens and infection of the whole eyeball and at last it leads to total blindness.

1. IODINE DEFICIENCY DISORDERS (IDD)

Iodine is an essential micronutrient for normal growth and development in animals and humans. Its deficiency not only causes goiter, but also is responsible for impaired brain development in the foetus and infant and retarded physical and psychomotor development in the child.

Prevalence

Children 6-<12 years

Goitre present	- 4.78%	
Grade I Goitre	- 4.66%	
Grade II Goitre	- 0.12%	
Deaf-mutism/Cretinism	- 0.18%	(ICMR, 2001)

Aetiology of Goitre

Environmental factors

(a) Environmental iodine deficiency The food crops grown on iodine-deficient soils will naturally be deficient in iodine and people subsisting solely on these foods will run the risk of developing IDD

(b) Goitrogens in food Certain chemical substances such as thiocyanates, thio-oxazolidone, flavanoids, disulphides, phenols, phthalates, biphenyls and lithium which are found in environment are called as goitrogens. Even excess of iodine is known to act as goitrogen

Intrinsic factors Some of the intrinsic factors such as failure to synthesis the thyroid hormone due to inherited and congenital defects in the hormone synthesis and secretion and peripheral resistance to thyroid hormone can also result in goitre

The spectrum of IDD

Depending on the stage of development, the iodine deficiency leads to a variety of disorder, which are

Foetus

Abortions

Stillbirths

Congenital anomalies

Increased prenatal mortality

Increased infant mortality

Neurological cretinism

Myxoedematous cretinism

Psychomotor defects

Neonatal goitre

Neonatal chemical hypothyroidism

Neonate

**Children and
Adolescents**

- : **Goitre**
 - Juvenile hypothyroidism
 - Impaired mental function
 - Retarded physical development

Adult

- : **Goitre with its complications**
 - Hypothyroidism
 - Impaired mental functions

Goitre Is defined as the non-neoplastic, non-inflammatory and non-toxic enlargement of thyroid gland. A thyroid gland whose lobes have a volume greater than the terminal pharynx of thumb of the subject examined will be considered goitrous. The prevalence of goiter is generally seen more among adolescents, young adults and schoolage children. More females are affected than males.

Cretinism: Endemic cretinism is associated with severe iodine deficiency during intrauterine life. A wide range of defects such as mental deficiency, deaf-mutism and spastic paralysis of legs in varying degrees are associated with this condition.

Clinically two types of cretinism are known.

(1) **Neurological cretinism:** Severe mental retardation, deaf-mutism, squint and spastic diplegia-spastic rigidity affecting the lower limbs leading to characteristic gait and brisk reflexes are the manifestations of neurological cretinism.

(2) **Myxoedematous cretinism:** These cretins exhibit signs of Hypothyroidism, i.e., coarse dry skin, swollen tongue, deep hoarse voice, apathy and mental deficiency. Associated skeletal growth retardation, weak abdominal muscle, sluggish bowel functions and delayed tendon reflexes are present in this condition.

Hypothyroidism: It is characterized by coarse and dry skin, husky voice, delayed tendon reflexes etc.

Psychomotor Defects: Child population from iodine-deficient areas shows poor scores in IQ tests, impaired school performance and poor motor coordination.

Impaired mental function: Population residing in iodine-deficient areas unusually exhibit reduced mental function, low intelligence levels and high degree of apathy, reflected in lack of initiative and decision making capacity of the people.

5. B Complex Deficiency

Most of B-complex vitamins are prosthetic groups for enzyme of biological importance. The B-complex vitamins which are water-soluble are easily excreted and do not have adequate stores in the body. Deficiencies of single B-vitamins are relatively rare, while multiple deficiencies often occur.

Aetiology

The major aetiological factors for B-complex vitamins deficiencies are

1) Inadequate intake

- Poverty
- Faulty cooking habits
- Losses due to storage, processing
- Bioavailability problems

2) Impaired absorption

- Chronic diarrhoea
- Malabsorption syndromes

3) Metabolic function

- Genetic abnormality (Inborn errors)
- Metabolic stress
- Drug - induced antagonism
- Increased urinary excretion
- Increased requirements

4) Depleted stores

- Poor nutritional status of mother
- Drug-induced depletion

Riboflavin deficiency

It is characterized by orolingual, dermal, haematological and ocular manifestation

Manifestations: Orolingual manifestations include soreness of tongue and burning of mouth and tongue. The lesions at the angles of mouth are termed as angular stomatitis. The lesions may progress to small cracks producing fissures and are covered by whitish or yellow crusts. The tongue is inflamed called glossitis. Dry chapped appearance of the lips with superficial ulcers termed, as cheilosis is present.

Folic acid and vitamin B₁₂ deficiency

Both folic acid and vitamin B₁₂ are involved in the synthesis of nucleic acids and amino acids. Folic acid deficiency in progress lead to megaloblastic anaemia. Vitamin B₁₂ deficiency commonly manifests as pernicious anaemia, which is genetic in nature and occurs due to deficiency of intrinsic factor in the gastric secretion. In adults, vitamin B₁₂ deficiency may result in neurological manifestations are peripheral neuritis, mental apathy and psychosis

Causes and consequences of Malnutrition in India

Malnutrition results from a combination of three key factors: inadequate food intake, illness and deleterious eating practices. In India, household food insecurity stems from inadequate employment and incomes seasonal migration, especially among the tribal populations, relatively high food prices, geographic and seasonal mal-distribution of food, poor social organization, and large family size (Edmundson, 1992)

A major determinant of protein energy malnutrition is household caloric inadequacy. According to National Sample Survey in 1993-94, the poorest 30 per cent of India's population consumed on an average, fewer than 1700 calories per day. At lower levels of caloric intake, people simply do not survive for long

While poverty largely explains the high level of malnutrition in India, additional factors are responsible for the concentration of the problem among women and children. Lack of information and education among women also underlies child malnourishment. Malnutrition directly or indirectly responsible for more than half of the deaths of children under 5 years of age worldwide. While India has successfully brought down infant mortality rate from 145 per 1000 live births in 1951 to 72 in 1996, most of the children who survive are malnourished. India's maternal mortality ratio of 420 per 100,000 live births is unacceptably high (ICMR, 2003)

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High levels of anaemia, low pregnancy weight gain, repeated acute infections, major chronic diseases, such as tuberculosis and inappropriate management of deliveries and important determinants of maternal and infant deaths. A large proportion of adult Indian women is at high risk of maternal mortality because their low pre-pregnancy height or weight may cause obstetrical difficulties. And when a malnourished or ill mother gives birth to a low birth-weight baby.

Malnutrition not only blights the lives of individuals and families, but also reduces the returns on the investment in education and acts as a major barrier to social and economic progress. Any deficiency during this period of rapid growth and development caused irreparable damage to the child and no subsequent attention may make up the loss. Improper development results in stunted physical growth and development, which lead to generalized functional disability, diminished productivity and inability to cope with environment hazards including resistance to infection (Harris, 1992). Good nutrition ensured protection while poor nutrition increased susceptibility to infections.

Effect of malnutrition

1. Direct effect

Deficiency of important nutrients will cause

- Protein energy malnutrition (PEM)
- Hypovitaminosis A
- Anaemia
- Goitre and cretinism

2. Indirect effect

- Malnutrition and infection interaction
- Low birth weight babies. This is due to the decrease in BMI status of expectant mothers
- High rate of child mortality
- High rate of maternal mortality

3. The over all effect

- Mortality and morbidity is high

- Reduced capacity for physical work
- Reduced mental efficiency

1. High mortality and morbidity

Mortality and morbidity rate is high due to infectious diseases like Measles, polio, whooping cough, Diphtheria, Tetanus, Tuberculosis, Diarrhoea and acute respiratory infections.

- Poverty
- Lack of immunization
- Low literacy level
- Lack of medical facilities

2. Reduced capacity for physical work

Malnutrition reduces the health of the population by it the capacity for physical work is also reduced

3. Reduced mental function

Malnourishment can also significantly lower cognitive development and learning achievement during the preschool and school years and subsequently results in low mental performance. According to the present stage of knowledge it appears that in children who had a severe PEM there is incomplete restoration of functional capacities of brain in spite of the intensive therapy resulting in apparent clinical case

Prevention of Malnutrition in India

Malnutrition is still a widespread health problem in India. Millions of children are still victims of protein energy malnutrition and lack of certain micronutrients, namely, iron, iodine and vitamin A in their diets. High Infant Mortality Rate (IMR), high levels of morbidity, nutrition related diseases temporary or irreversible disabilities and low productivity are some of the outcomes of malnutrition. The effects of malnutrition are particularly evident among the very young children, expectant and nursing mothers (Devadas, 1993)

Diet surveys carried out in India have shown that the diets consumed by a large majority of the vulnerable groups of the population are inadequate in quantity and quality. A fair section of the population does not get enough food to eat. Undernutrition and malnutrition are widely prevalent among the low-income groups.

Food requirements: The most urgent necessity is the increased production of cereals. This will help to prevent undernutrition among the low-income groups. Because it is difficult to produce adequate amount of milk, meat and eggs due to low percaput land availability, attempts should be made to increase the production of cereals, pulses, groundnut and green leafy vegetables.

Control of population growth: Population in India is rapidly growing at the rate of 2.5% per annum. If the population increase is not controlled, it will be difficult to provide adequate amounts of even cereals to the people. So it is also leads to malnutrition.

Nutrition education Education on the nutritive value of the locally available foods like cereals, pulses and leafy vegetables and formulation of low cost balanced diet based on cereals, pulses, nuts and green leafy vegetables for feeding the vulnerable groups

Increased income: Inadequate income is another important cause for the prevalence of malnutrition. Steps should be taken to increase the wages and to supply the essential foods at controlled rates to the low-income groups as a part of the wages

Improvement of sanitation: Diseases due to infection and infestations by microorganisms and parasites are important factors involved in the causation of malnutrition. There fore it is essential to improve the hygienic and sanitary conditions of the surroundings in the locality.

Encouraging kitchen gardens and poultry farming: Production of protective foods such as green leafy vegetables and eggs can be stepped up by encouraging kitchen gardens and poultry units on a cottage scale. This will help to provide protective foods to the family. A part of the produce can be sold, thus providing additional income to the family (Swaminathan, 1986)

Nutrition Programmes in India

Nutrition interventions aimed at provision of food and nutrients directly to people who are at risk of developing malnutrition constitute a families strategy pursued by the health and social welfare sectors in many developing countries. The under laying principle of this approach is to supply the deficient/missing nutrient either through food which is habitually consumed (food fortification) or through its

administration in medicinal form (supplementation) at frequent intervals. This approach can hardly be expected to eradicate the root causes of malnutrition namely poverty, ignorance and inadequate health care. For this several nutrition intervention programmes that are currently in operation, either singly as vertical programmes, or as a part of health and welfare services (integrated programme)(NIN, 2000).

The programmes:

Two specific kinds of programmes

1) Food supply and supplementation programmes

- Supplementary feeding programme (SFP)
- Mid-day Meal Programme (MDM)
- Public distribution system (PDS)
- Food for work programme (FFW)

2) Vitamins and mineral supplementation programme

1) Food supply and supplementation programmes

Supplementary feeding programmes or special nutrition programme (SNP)

The Government of India in 1970-71 initiated a supplementary feeding programme for preschool children in different state. Initially, the programme included only preschool children as beneficiaries but subsequently it included pregnant and lactating women. This is a ready-to-eat food supplementation programme. The results of two carefully conducted studies formed the scientific base of this programme

i) Food intake studies undertaken as part of a comprehensive nutrition survey of preschool children (1-5 years) conducted by NIN, showed for the first time that the diets of preschool children are primarily deficient in energy content and not protein as hitherto believed and that the extent of deficit is of the order of about 300 kcal/child/day

ii) Based on this finding, a long-term feeding trial involving about 400 preschool children, living in villages were given food supplements providing daily about 300 kcal and 3 g protein per Childs six days in a week for 14 months under supervision.

The results of this study demonstrated that making good the energy deficit in the diets of preschool children had not only prevented occurrence of Kwashiorkor/marasmus

and severe degree of growth retardation, but also promoted their growth at rates comparable to those seen in children of well-to do communities.

The programme is sponsored by the ministry of Human Resource Development and implemented by the Department of Women and child welfare. The population of urban slums, tribal areas and drought prone rural regions are covered on a priority basis. The emphasis in the programme, is on making use of locally available inexpensive foods like cereal / millet and pulse mixes to keep the programme overheads to the minimum and also to achieve a high degree of programme acceptance.

This SNP has now been integrated to the ongoing ICDS programme and is delivered as a part of package of primary health care education services.

Recipients	Calories	Grams of Protein
Children up to 6 Years	300	8-10
Adolescent Girls	500	20-25
Pregnant and nursing mothers	500	20-25

2) Mid-day Meal Programme (MDM)

Under applied nutrition programmes (AND), which flourished during the 1960s and 1970s, the MDM programme received great impetus. The programme in principle, envisages supplying on supplementary meal providing a third of daily calorie requirement and 50% of protein needs of the child. This programme comes under Ministry of Education. On the recommendation of the National School Health Committee, the Government of India started a scheme for providing mid-day meal to school children in all states with effect from 15th August 1995. The meals are given

based on a combination of cereals, pulses and leafy vegetables. Eggs are given once a week. Such diet would increase the amount of vitamins and minerals as result in weight gain and clearance of deficiency symptoms.

3) Food For Work (FFW) Programme

Food for work (FFW) programme, subsequently designated as National Rural Employment Programme (NREP), and Jawahar Rojgan Yojana (JRY) are important. The purpose of this approach is to provide firstly, the work to rural unskilled labour during lean agricultural period and secondly, to supply food grains as part of wage. Under this programme special type of works like construction of roads, school buildings, dispensaries, excavation of water tanks, reservoirs, canals etc.

The programme did help in creating several million mandays of work but the drawbacks, which included

- Lack of managerial support to plan suitable works and supervise their implementation
- High costs of the projects
- Frequent interruptions in supply of food grains
- Inferior quality of food grains

There are resulted in undue delay in completing the works

4) Public Distribution System (PDS)

The Public Distribution System is the major food-subsidy/income transfer programme in the country, administered by the ministry of Food and Agriculture. It is another approach to meet the basic food (nutrients) needs of the poor through a network of Fair Price Shops (FPS). The commodities given by this programme are, wheat, rice, sugar and kerosene

It has been inadequately targeted, with a larger number of beneficiaries actually coming from non-poor house holds. In poorer states, they take less than their share of supplies from PDS mainly because of a weak administrative capacity and the inability to move the food stocks

VITAMIN AND MINERAL SUPPLEMENTATION PROGRAMME

1) The National Vitamin A Prophylaxis Programme

Initiated by the Government of India in 1970. In this each preschool child (1-3 years) receives a massive dose of 200,000 IU of vitamin A prepared in oil and administered orally once in every six months for preventing the vitamin A deficiency disorders.

The programme is operated through the existing health and ICDS infrastructure by auxiliary nurse midwives (ANMs) and Anganwadi workers (AWWs).

This programme is covering a total of about 30 million children.

2) Alternative Approaches for Delivering Vitamin A

Universal Programme of Immunization (UPI)

Linkage with expanded programme of immunization and horticultural development

Vitamin A with the expanded programme of immunization now being called as Universal Programme of Immunization (UPI)

The first dose - 100,000 IU of vitamin A - infants along with measles vaccination

Second dose - 200,000 IU at 18 months age along with booster

This linked approach has been criticized on apprehension that this may perpetuate continued reliance on synthetic vitamin A as an integral part of immunization schedule and detract the people from consumption of vitamin A rich foods particularly carotene-rich fruits, vegetables etc. The only way to prevent this, to mount an appropriate nutrition/health education programme particularly women while delivering a host of other MCH and family welfare services. Every contact is a golden opportunity for the worker to carry the message home. Horticulture development programmes by the agricultural extension workers are encouraging people to grow carotene - rich foods. A nutrition orientation to horticultural efforts and horticultural orientation to health/nutritional measures is essential in achieving the objective of increasing vitamin A intake of children through dietaries.

3) National Nutritional Anaemia Prophylaxis Programme (NNAPP)

Government of India launched this programme in 1970. Under the programme, women (pregnant, lactating and family planning acceptors) receive tablets which contain 60 mg of elemental iron and 500 µg of folic acid per tablet (folifer) while the children under 12 years receive tablets containing 20 mg of iron and 100 µg of folic acid per tablet for very young children, who cannot swallow tablets liquid preparations are preferred. The distribution is undertaken by the infrastructure provided at primary health centres, sub-centres, MCH centres and ICDS 'anganwadis'.

National Iodine Deficiency Disorder Control Programme (NIDDCP)

Studies conducted by All India Institute of Medical Sciences (AIIMS), showed that iodized salt distribution drastically reduces the goitre prevalence, the Government of India, initiated the National Goitre Control Programme (NGCP) in 1962

Objectives of these programmes are

- To identify and assess the prevalence of goitre in suspected areas through systematic surveys
- To prevent and control goitre through production and supply of iodised salt in place of common salt in endemic areas
- To assess the impact of the programme by undertaking re-evaluation of the areas after 5 years of continuous supply of iodised salt

The programme has now been redesigned as National Iodine Deficiency Disorder Control Programme (NIDDCP)

A review of the 30 year old programme, undertaken by nutrition foundation of India (NFI) in 1983 suggested that the problem of goitre is not only more widespread and its severity is more intense. The failure was more due to operational and administrative reasons than the technology. Main issues, as seen in other nutrition action programme, were non-availability of the basic nutrient input to match the requirement. In addition, the transport difficulties, cost factor, weak enforcement of provisions of PFA in respect of iodised salt, lack of supervision, absence of quality-control measures accounted for the failure of the programme (Vir, 1994).

New initiative: Realising the serious dimension of the problem the Government of India has taken new initiatives.

a) Iodization of the entire salt used for human consumption in the country (Universal Iodization). The programme was included in the 7th five-year plan with an outlay of Rs 210 millions.

b) Liberation of the production of iodized salt (was the basic constraint in the programme) by giving permission to private sectors to produce it and subsidizing the cost of production by providing financial assistance for setting up the plants.

c) High priority 'B' for allocation of Railway (covered) wagons to transport iodised salt.

d) Free technical assistance by the salt department for installation of plants and training of operations/ technicians on quality control.

As a result of this, the iodized salt production, which was almost static. Organisational changes have been effected to ensure nation-wide monitoring system to carry out quality control of the product at manufacturing, storage and consumption levels to ensure that the salt contains at least 15 ppm of Iodine at consumer level.

Organization In the context of IDD control programme the ministry of Health and family welfare is the policy making body of the Government of India. The salt Department under the Ministry of Industry is specified as nodal agency for monitoring production, distribution, quality control and subsidy payment to the iodised salt manufactures. The Directorate General of Health Services (DGHS) under the Ministry of Health and Family welfare is responsible for co-ordinating with the state level units. The State Governments are responsible for the distribution of the iodised salt within the state either through public distribution system or through open market. The Director of Civil Supplies of each state issues notification-banning sale of non-iodized salt for edible purpose in the goitre endemic areas.

Integrated Child Development Services (ICDS) Programme

The programme started on 2nd October 1975 by UNICEF. It is implemented by the department of women and child development at the central level in co-ordination with ministry of health. In the state level implementation either by the

department of social welfare/ women and child development health or a separate directorate of ICDS.

The specific objectives:

- i) To improve the nutritional and health status of children on the age group of 0-6 years and adolescents.
- ii) Lay the foundation for proper psychological, physical, and social development of the child.
- iii) Reduce the incidence of mortality, morbidity, malnutrition and school drop out.
- iv) Achieve effective co-ordination of policy and implementation amongst the various departments of promote child development.
- v) Enhance the capability of the mother to look after the health and nutritional needs of the child through proper nutrition and health education (Sarma, 1993).

Components of service activities of ICDS

The packages of services are the important component of this programme. The focal point of the convergence of these services is the 'anganwadi' (AW), a preschool child centre, located within the urban slum or village. Each centre is managed by a lady worker called 'anganwadi workers' (AWW) is variably assisted by another lady 'helper'. The services include:

1) Supplementary nutrition

This is one of the major components of ICDS. Cereals and pulses from essential ingredients are cooked into a recipe, which can be salty or sweet preparation. In some states a ready to eat snack containing some basic ingredients is produced. 300 kcal of energy and 15 g of protein are supplied to children through ICDS. Double the daily supplement is provided to the severely malnourished children.

2) Immunization

Launched in 1978. All infants in the project area are immunized against infectious disease such as tuberculosis, diphtheria, whooping cough, tetanus, and poliomyelitis. Measles vaccination also provided. All pregnant women are immunized against tetanus.

3) Health check up treatment of minor ailments and referral services

At the anganwadi, children, adolescent girls and pregnant and lactating mothers are examined and treated by the local visiting health personnel, such as the Lady Health Visitor (LHV) and Health Assistant and multi purpose Worker (MPW). It provide link between the village and Primary Health Centre and sub-centres.

4) Growth monitoring

It is done with the help of special growth charts. From these charts, nutritional status of each individual can be assessed periodically.

5) Non-formal educational activities

The main purpose of the preschool education component, particularly for those between 3 and 5 years is to stimulate and satisfy the curiosity of the child rather than follow any rigid learning curriculum (Rao, 1996).

6) Vitamin A prophylaxis programme

Every child will get an oral dose of 2,00,000 IU of vitamin A every six months through this programme

Extended scope of ICDS

According to recent information from the Department of Women and Child Development (DWCD), the infrastructure of the ICDS is now being used to introduce newer interventions meant for development of adolescent girls

National Diarrhoeal Control Programme

Launched in 1981 to reduce mortality in under five due to diarrhoea through the introduction of Oral Rehydration Therapy (ORT). ORT based on the administration of correct oral fluids while allowing food intake, provides a balanced water and electrolyte replacement at low cost and saves lives

National Vector Borne Disease Control Programme

Malaria has been a major health problem in India. Majority of the infections were due to plasmodium falciparum and P. vivax. For controlling this disease the Government of India launched a programme - National Malaria Control Programme (NMCP) in 1953 and it is renamed as National vector Borne Disease Control Programme in 2003

Combating vitamin A deficiency and anaemia

The role of the Ministry of Food Government of India

The food and nutrition Board of the Department of Food Undertakes three basic projects to improve the nutritional status of the people.

1) Nutrition education

2) Development and promotion of nutritious food and

3) Fortification and enrichment of foods

1) To impart nutrition education and training, an important tool for nutrition promotion, the Department has, in collaboration with State Governments

→ Set up mobile food and nutrition extension units (NEUs, with audio-visual aids and trained personnel to organise live demonstrations of various aspects of food and health in all parts of the country

→ Established food and nutrition extension centres (FNECs) to encourage people to preserve fruits and vegetables within the home, particularly during the glut season

→ Launched an integrated nutrition education scheme to equip grass root workers at all levels with knowledge of food, nutrition and health, so that this is disseminated to the community

→ Celebrated national nutrition weeks to intensify nutrition awareness among the masses

→ Established food processing and nutrition centres to provide demonstration/training on the preservation of foods

2) Under the second project, nutritious foods such as miltone, RTE extruded foods, and energy foods are developed

3) The third project undertakes the fortification of milk with vitamin A and salt with iron, in collaboration with state and voluntary agencies. These are economical and effective means of preventing nutritional blindness due to vitamin A deficiency and iron deficiency anaemia (Rao, 1991)

Conclusion

Most of the deficiency diseases are not killing diseases unless they take a serious form. But they cause physical and mental debilitation which determine health and efficiency and which results in constant misery and suffering and poor diet. Malnutrition is like the iceberg in an ocean, only its tip being visible. The malnutrition crisis is a vicious spiral. Under development means low incomes relatively poor health, high infant mortality, poor diet and malnutrition. The nutritional deficiency diseases should not be considered either a medical or clinical problem or lack of medical knowledge but it is related to the social and economic causes and the prevailing economic system. Malnutrition, the worst affected are our children who must become able citizens of tomorrow in order to make our country one of the foremost in the world. Various nutrition programmes have been launched over the years therefore at least the tip of the iceberg has nearly melted.

Discussion

1) What are goitrogens? Examples

Goitrogens are chemical substances are known to interfere with iodine metabolism at various levels and it leads to goitre examples are thiocyanates, thio-oxazolidone, flavanoids etc. Even excess of iodine is known to act as goitrogen.

2) If any harmful effects caused when iodine salt is daily taken?

If excess iodine is also acts as a goitrogen so it leads to goitre. But in our body only enough quantity of iodine is absorbed and excess iodine is excreted through urine

3) Nutrition week?

September first week

4) Present status of the Nutrition Programme - A cup of milk and a shake hand?

Now it is not properly working. Because of the improper launching of the programme

5) Role of Home Scientists in Nutritional Programme?

Home scientists, at first they have to identify the nutrition status of the people. Educating the people on health and hygiene and to give various cooking practices to enrich the nutrients in the food by formulation of nutritious recipes. To enhance the food production and preservation

6) If ICDS programme is properly working in Kerala?

Yes, in Kerala it is working properly through Anganwadi centres.

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ABSTRACT

Nutrition is a major factor in bringing out the maximum potentiality that one is endowed with, both physically and mentally. Good nutrition depends upon adequate food supply and this in turn on a sound agricultural policy and a good system of food distribution. Malnutrition is an ecological problem that doesn't occur alone. Although the term malnutrition includes both under nutrition and over nutrition, in developing countries, under nutrition is the major public health problem.

In India like developing countries the major nutritional problems of public health importance are Protein Energy Malnutrition (PEM); Nutritional Anaemia; Vitamin A Deficiency (VAD), Iodine Deficiency Disorders (IDD) and B complex Deficiency (UNICEF, 1992) The most vulnerable groups of these deficiency diseases are pre school children, adolescents, pregnant and lactating women, lower income group and population living in tribal tracts. Inadequate food intake, illness and ignorance are the three key factors resulting in malnutrition.

In Protein Energy Malnutrition, the children embrace at one end of the spectrum, the puffed up cases of kwashiorkor, the shrivelled cases of marasmus and as the other, cases of nutritional dwarfing. In between these extremes are various degrees of PEM (Joshi, 2002). In India the prevalence of kwashiorkor is 0.04 per cent and marasmus is 0.07 per cent (ICMR, 2001). Though keratomalacia (nutritional blindness) has significantly declined as a public health problem, conjunctival xerosis (and Bitot's spot) still remains a problem of public health scale not only among preschool children but among older children as well. Even mild vitamin A deficiency increases morbidity and mortality in children, highlighting the public health importance of this disorder. ICMR (2001) had reported that in India the overall prevalence rate of Bitot's spot in children is 0.70 per cent and night blindness is 0.71 per cent and night blindness in pregnant women is 2.76 per cent. Anaemia is another public health problem of significance, which has an impact on physiological and physical development, behaviour and work performance. It affects mainly women in the reproductive age group and young children. The prevalence of anaemia in pregnant women is 85 per cent, in that 10 per cent is severe case, in children it is 74

per cent, in adolescent girls it is 60-70 per cent and in men it is 38 per cent. Goitre and cretinism are the clinical features of IDD (Iodine Deficiency Disorder). Cretinism is seen in children and goitre in all age groups. The prevalence of goitre in children is 4.78 per cent and in adults it is 5 per cent (ICMR, 2001). B complex deficiency especially riboflavin and folic acid deficiency occur along with other deficiency diseases.

Prevention and control strategies thus assume great importance in the context of national economy and development. Government of India started several intervention programmes in the last 3-4 decades including National Goitre Control Programme (1962) which was renamed as National Iodine Deficiency Disorder Control Programme (NIDDCP) in 1992, National Nutritional Anaemia Prophylaxis Programme (1970) which was renamed as National Nutritional Anaemia Control Programme (1996), The National Vitamin A Prophylaxis Programme, The Mid day Meal Programme (1970) and ICDS programme

The Integrated Child Development Services (ICDS) Programme of the Department of Women and Child Development was started in 1975. It provides a package of services which cover almost all the intervention programmes of the government and the focal point of the convergence of these services is the anganwadi, a preschool child centre. (Rao, 2003)

ACTIVE PACKAGING

INTRODUCTION

India is the second largest producer of fruits and vegetables in the world. The production of fruits is estimated to be 46 million metric tonnes and that of vegetables is 91 million metric tonnes. Unfortunately, unlike other horticulturally rich countries, Indians do not get the basic daily requirement of fruits and vegetables. High rate of population growth further offsets the higher production of fruits and vegetables. In addition, there is a considerable gap between the gross production and net availability of fresh fruits and vegetables and their products due to heavy post harvest losses. Furthermore only 1.3% of the whole production is commercially processed (Anon, 1996). India exported 0.54 million tonnes of fresh fruits and vegetables and 0.19 million tonnes of processed fruits and vegetables, compared to many countries in the world whose position is much below India's production level (Anon, 1996). Therefore, in order to satisfy the demand of fruits and vegetables by the ever increasing population as well as meeting the requirement of the processing industry and export trade, only increasing the production and productivity will not be enough. A lot more emphasis need to be given to postharvest management and packaging of fruits and vegetables and their products.

IMPORTANCE OF PACKAGING

The increased production of fruits and vegetables will have significance only when they reach the consumer in good condition at a reasonable price. The existing post harvest losses of fruit and vegetable could be considerably reduced by adoption improved packaging, handling and efficient system of transport (Roy *et al.*, 2000).

Packaging of fruits and vegetables is undertaken primarily to assemble the produce in convenient units for marketing and distribution. The food package serves a number of purposes. The package protects the product from tampering, maintains quality and retains freshness. The package also establishes an identity for the product in the market place which allows companies to enhance brand loyalty. The package must be capable of

- 1.) protecting the produce from the hazards of transport

- 2.) preventing microbial and insect damage
- 3.) minimising physiological and biochemical changes.

The important considerations in selecting the packaging material are:

- 1.) The product itself
- 2.) The systems of production
- 3.) The systems of storage
- 4.) The systems of handling
- 5.) The systems of transport
- 6.) The system of merchandising
- 7.) The consumer attitude
- 8.) The requirement of recycling/ reuse/ disposal etc. (Roy *et al.*, 2000)

Packaging designs:

The traditional types of food packaging are boxes, cartons, metal cans, glass bottles and jars. The new innovative packaging designs are aseptic packaging, PET containers, microwaveable containers, controlled/modified atmosphere packaging and active packaging.

ACTIVE PACKAGING:

Active packaging is termed as the dawn chorus of the advent of modern era of packaging. It is also known as Intelligent packaging, Innovative packaging, High-tech packaging, Smart packaging, etc.

The term 'Active Packaging' was introduced by LABUZA in 1987. Simply, packaging may be termed 'active' when it performs some role other than providing an inert barrier to external conditions.

Active packaging is a group of technologies in which the package is actively involved with food products or interacts with internal atmosphere to extend shelf life while maintaining quality and safety (Floros *et al.*, 1997).

According to Brody *et al.*, (2001), Active packaging, sometimes referred to as "interactive" or "smart" packaging, is intended to sense internal or external environment change and to respond by changing its own properties or attributes and hence the internal packaging environment.

Active packaging is also called as intelligent system in the view that it senses and measures variations in the environment of the package and also communicates the same to an observer (Brody, 2000). ✓

SCOPE:

Active packaging was developed as a series of responses to unrelated problems in maintaining the quality and safety of foods. Some properties of foods which can be addressed by active packaging are to sustain life in foods, prevent oxidative attack on food constituents, retain flavour and for facilitating the serving of the food for consumption.

In India active packaging could be highly useful and potent system for extending the shelf life quality of a wide range of traditional dairy products, such as paneer, khoa, and sweetmeats etc. In addition to various cheeses which are growing very fast in our country (Prabha *et al.*, 2003).

Active packaging refers to the incorporation of certain active components in packaging films or in the form of inserts that interact with food environment within packaging containers. Other active packaging systems use antimicrobials, antioxidants, enzyme inhibitors, stabilizers, flavour absorbers, emitters, light blockers/regulators, antifogging and anti-tackling agents and temperature sensing or controlling systems (Floros *et al.*, 1997).

The principles of active packaging are adsorption, absorption, porosity control, polymer permeability, thermal expansion, hydrolysis, acid base reaction, inorganic/organic oxidation, gas scavengers and gas emitters of any food products so packaged.

Potential Technologies being used in Active Packaging:-

1) OXYGEN SCAVENGING:

Residual oxygen in food packages accelerates the spoilage of many foods. Oxygen can cause offodour development, colour change, nutrient loss and microbial

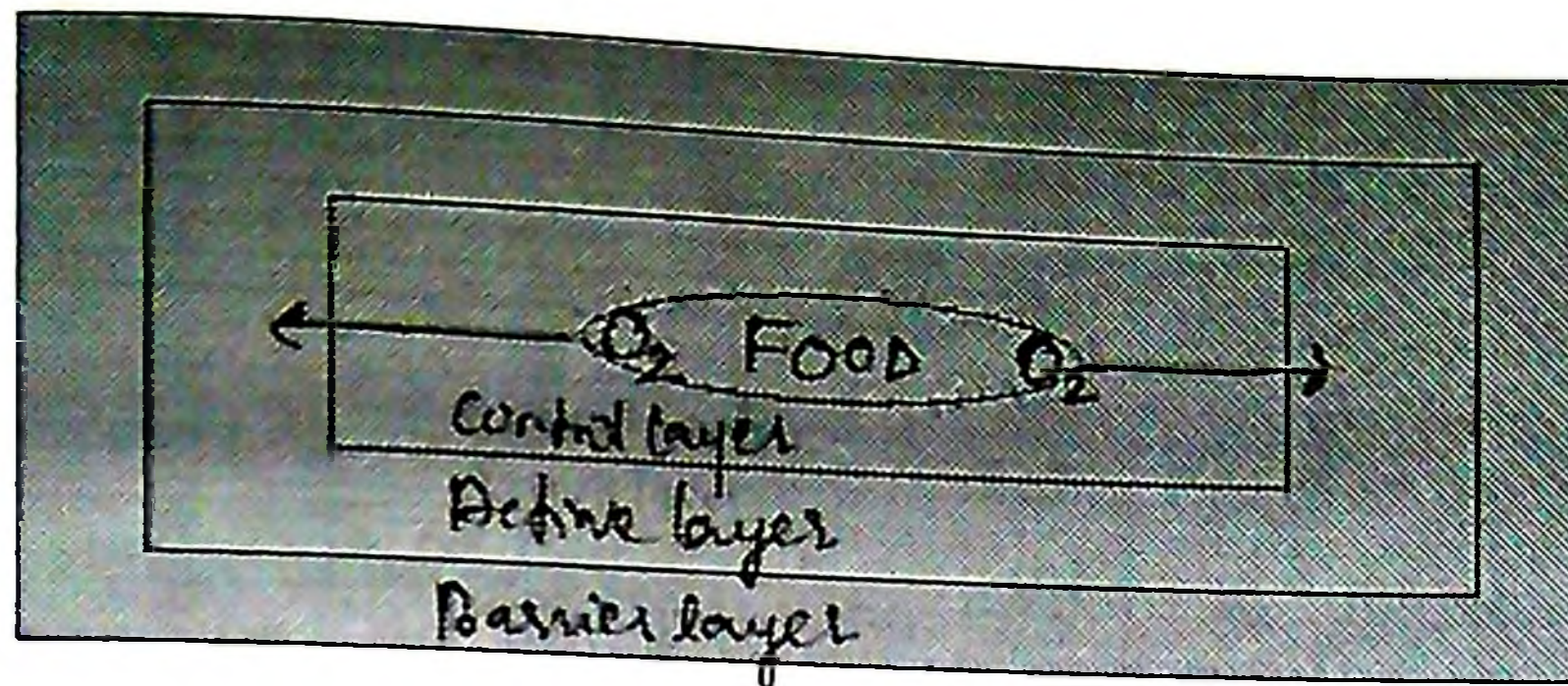
attack. Various techniques such as absorption, interception and scavenging has been employed for removal of oxygen. However most of the work has been done in incorporation of oxygen scavengers especially into plastics such as multilayer films and PET bottles (Brody *et al.*, 2001). The most widely researched and patented area of active packaging is the use of oxygen absorbing systems. (Rooney, 1995, Floros *et al.*, 1997).

Studies by Powers and Berkowitz (1990) at the US army Natick Research, showed that a Fresh Pax Oxygen scavenger enclosed in a high gas barrier pouch made of polyester/Aluminium foil/HDP with baked meal, ready to eat bread prevented mold growth on bread for 13 months at ambient storage temperature. Alarcon and Hotchkiss (1993) have also reported prevention of mold growth and rancidity in preservative free white bread and low moisture, low fat mozzarella cheese for eight weeks at refrigerated or ambient temperature. Ravinder kumar *et al.*, (1997) observed that packaging of peda in a multilayer pouch EVA/DVDC/EVA containing oxygen scavenger sachet and indicator tablets for extending shelf life upto two months at 37°C, five months at ambient temperature and six months at 20°C whereas packaging under MAP conditions resulted in shelf life of 15 days both at 37°C and ambient temperature and 30 days at 20°C.

CSIRO Division of Food Processing, Australia has developed packaging films containing photo sensitive dye and an excited Oxygen trap. On illumination of films, dye molecules become excited and pass on their excitation energy to oxygen from package headspace, these excited oxygen molecules react with trap and the process continues until all oxygen is trapped.

Mechanism of Oxygen absorbing films

Active material may contain multiple layers with each layer having different functions. The inner layer is permeable to oxygen. Outer layer will be high oxygen barrier. There will be central layer present in the multilayer structure to minimize any migration of oxygen absorber in the food. Many absorbers describing plastics containing oxygen absorbers have been reviewed (Rooney, 1995). Finally the oxygen absorbing compounds have been added to labels that can be directly affixed to the interior of a package.



Limitations of Oxygen absorbers:

There are however many limitations in application of oxygen absorbers. It causes undesirable appearance in flexible package. Sachets form of oxygen absorbers is not well perceived by consumers. It increases the cost of the product by approximately 2.5% to 10% which may be prohibitive for products with low profit margin. There are also chances of developing anaerobic bacteria such as *Clostridium spp.* or facultative anaerobic bacteria such as *Listeria spp.* There is also danger of accidental consumption of absorbing compounds either directly from sachet or due to migration out of the film (Gnanasekharan & Floros, 1997)

2) GAS PERMEABILITY CONTROL

Oxygen Control

Fresh and minimally processed foods are often intentionally packaged in reduced oxygen environment to decrease enzymatic, biochemical and aerobic microbiological activities. But when oxygen concentration inside the package reduces to zero level, anaerobic respiration produces odoriferous aldehydes, ketones and some toxins are produced in case of low acid foods. To overcome this, oxygen permeable package materials have been developed. Some packaging materials with adjuncts that are sensitive to temperature and significantly alter their gas permeabilities and packaging films containing pores filled with waxes which melt and flow out of the pores when temperature increases, are in use. Landec's (Intellipac™) technologies are probably the widely known temperature responsive package materials which are based on a side

crystallisable polymer. Oxygen permeation rates of 18.06 m²/day and carbon dioxide permeation rates of 7.74 m²/day are achievable and effective at various controllable ratios for high respiration rate, fresh produce such as broccoli, mushrooms, asparagus and strawberries.

Carbondioxide control:

It is a complimentary approach to oxygen control. High Carbon dioxide levels ie 10-80% are desirable for some foods such as meat and poultry. This inhibits surface microbial growth and thereby extends shelf-life. A mixture of Iron and Ca(OH)₂ in the form of sachet, which scavenges both oxygen and carbon dioxide, has been used to package fresh ground coffee in flexible bags. This sachet protects the flavour of the product while preventing carbon dioxide built up in the package. Another application of carbon dioxide absorbers is to absorb the carbondioxide produced during ripening and MAP.

3.ANTIMICROBIAL PACKAGING:

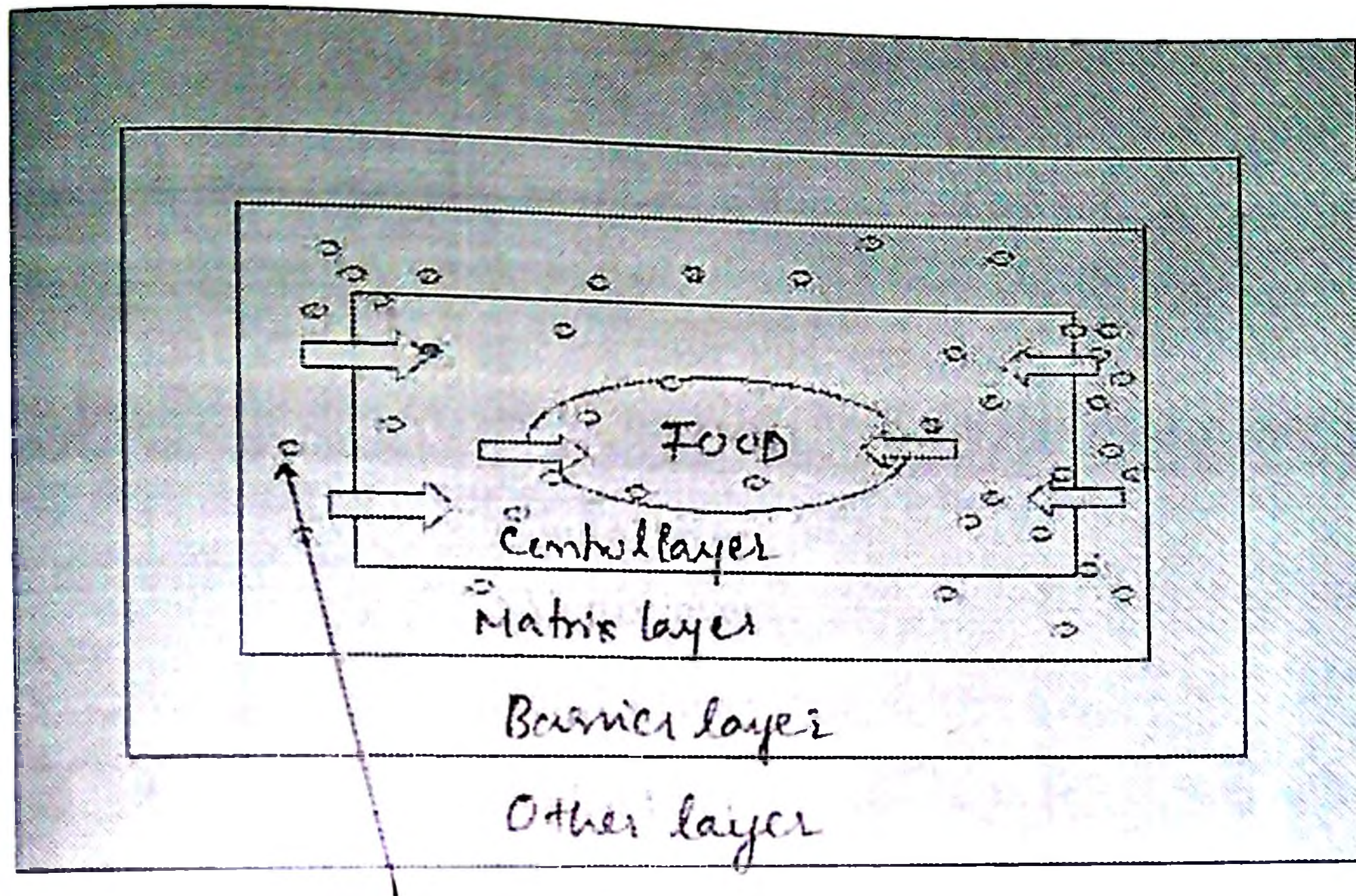
Microbiological growth is one of major mode of deterioration of foods. It is primarily the growth of pathogenic bacteria and mycotoxin producing mold that must be inhibited to ensure a safe product. Antimicrobial packaging is such a component of active packaging which involves the incorporation of antimicrobial agents directly into packaging film, that contact the surface of the food.

The antimicrobial substances incorporated into package materials can control microbial contamination by reducing growth rate and maximum growth population and extending the lag period of the target microorganisms (Han & Floros, 1997)

Structure of an Antimicrobial releasing Active packaging film

Packaging materials containing antimicrobial compounds require a multilayer structure to achieve controlled release (Floros *et al.*, 2000). Multilayer films may be composed of (a) a matrix layer containing the active compound, (b) a control layer with known diffusivity to control the migration of active compounds into the food surface and

(c) a barrier layer to prevent migration of active compounds towards the outside of the package and provide appropriate barrier properties.



Antimicrobial substance

Compounds used for Antimicrobial packaging

Ideal antimicrobial packaging material would have broad spectrum activity, be active at low concentration, demonstrate no adverse sensory effects and be low in cost

Antimicrobial agents to be used in food packaging:

CLASS	EXAMPLES
Organic acids	Propionic, benzoic, sorbic acids
Bacteriosins	Nisin
Spice extracts	Thymol, p-cymene
Thiosulphates	Allicin
Enzymes	Peroxidases, lysozymes
Proteins	Conalbumin

Isothiocyanates	Allylisothiocyanate
Antibiotics	Imazilil
Fungicides	Benomyl
Chelating agents	EDTA
Metals	Silver
Parabens	Heptyparaben

(Hotchkiss, 1995)

Most widely publicised antimicrobials are silver salts on zeolite incorporated in plastic films and sheets or on material surfaces and into purge absorbent pads for fresh meat products

As the first step in the development of antimicrobial packaging materials for preservation of meat products, the efficacy of several approved food grade antibacterial agents (fatty acids and essential oils) to control the growth of meat spoilage organisms has been assessed. The fatty acids, palmitic and lauric acids showed the highest levels of inhibition with minimum inhibitory concentration of 500 and 250 µg/ml, respectively. Oleic, stearic, myristic and palmitic acids were completely ineffective. The most effective essential oils were pimento, cinnamon and clove and rosemary, atleast 5 species of the bacteria tested were inhibited by a 1/100 dilution of these oils (Quattara *et al.*, 1997)

Sorbic acid and its potassium salts have been studied as preservatives for the packaging of these products. They were incorporated into wax layer for natural cheese, wet wax coating on packaging paper and edible protein coating on intermediate moisture foods. The potassium sorbate concentration on the cheese surface is critical and must be maintained above 0.1% to inhibit mould growth. Processed American cheese was packaged with 40µm thick HDPE films containing 10% sorbate initially and stored at room temperature, the surface concentration was maintained above 0.1% and such packaging enabled the cheese to be microbe free for five months at room temperature

Benzoic anhydride incorporated into LDPE films exhibits strong antimycotic activity when in contact with microbiological media and cheese (Hotchkiss, 1995). LDPE

films containing 0.5–2% benzoic anhydride delays mold growth on cheese. Antimicrobial enzymes such as glucose oxidase, which forms hydrogen peroxidase and alcohol oxidase have been bound to the inner surface of food contact films such as alginate films, which can be used to preserve fish.

Japan Freund Industrial Co. Ltd has patented the process of microencapsulation of ethanol in specially engineered silica containing a minimum amount of 55% ethanol by weight and trademarked as ETHICAP. According to Black *et al.*, 1993, the packaging incorporated with Ethicap doubled the shelf life of pita bread. Ethicap has also proved effective in controlling spoilage by *Saccharomyces cerevisiae* in gas packaged strawberry and vanilla layer cakes and cherry cream cheese cake. Seiler and Russel (1993) also reported that ethanol at a level of 1% by product weight, delayed the onset of ropiness caused by growth of *Bacillus subtilis*. They also reported that low concentration of ethanol (0.5%–1.0% by weight) inhibited bacterial growth in whipping cream and custard, two well known vectors of food poisoning bacteria in filled bakery products. These studies clearly illustrate the antibacterial properties of ethanol when incorporated directly into media or a food product.

In recent years, the FDA has approved the use of chlorine in packaging system to release chlorine dioxide. Food Science Australia is developing systems to release sulphur dioxide to control mould growth on contained fruits.

4) ETHYLENE CONTROL.

Ethylene is a hormone, which accelerates ripening in fruits followed by senescence. Removal of ethylene from plant environment can significantly retard post harvest catabolic activity in fresh produces and complement modified atmosphere preservation process.

Ethylene removal media are usually composed of pellets of porous solids such as activated alumina, vermiculite and silica gel that have been impregnated with KMnO_4 . These solids act as the absorptive surface to trap the gas molecules and carrier for permanganate, which oxidises ethylene to ethyleneglycol.

Packaging is incorporated with ethylene scavengers in several different forms including blanket, tubes and sachets. Sachets are used in individual boxes of fruits and

vegetables, whereas blankets and tubes are commonly used in transport vehicles. Friss pack is a paper incorporated with KMnO_4 for use in corrugated fiberboard cases manufactured by Dunapack, Hungary. Activated charcoal impregnated with a palladium catalyst and placed in proper sachets effectively removes ethylene by oxidation from packages of minimally processed kiwi, banana, broccoli and spinach.

Good quality green bananas were treated with ethylene and packaged in LDPE bags, the ethylene being immobilized with synthetic zeolite. The bananas were stored at 15°C for 30 days, followed by 20 days at 18°C . It was concluded that bananas packaged in LDPE and stored at 15°C enabled storage in a mature condition for long periods and retained the optimum sensory characteristics for 5-7 days. Anuradha and Siddiqui (1998) studied the effects of post harvest treatments, MAP and MAP with KMnO_4 impregnated on chalk sticks on storage life of ber (*Zizyphus mauritiana*) fruits. Fruits were stored under ambient temperature $33 \pm 4^\circ\text{C}$ and relative humidity $68 \pm 5\%$ for 6 days. Results suggested that MAP with KMnO_4 was more effective than hot water treatment in prolonging shelf life of ber fruits by reducing rate of ethylene production and ripening related processes over the 6 day period.

(5) MOISTURE CONTROL

Since the metabolism of fats and carbohydrates produces water, condensation or sweating is a problem in many kinds of packaged foods particularly, fresh fruits and vegetables or minimally processed prepared foods. This condensation moistens the product surface, soluble nutrients leak into the water encouraging rapid growth of ubiquitous mould spores and leading to the loss of nutrients from the product. Clays, molecular sieves and silica gel have been used to protect sensitive products against water and humidity. To date, silica gel has been the moisture absorbent of choice for dry foods which can absorb upto 35% of their own weight and maintain water activity well below 0.2. Molecular sieves such as zeolite can absorb water (upto 24% of their own weight) and off odours. Moreover, the super absorbent polymers have been shown to absorb water upto 500 times their own weight (Rooney, 1995). Use of humectants between two highly permeable layers of a plastic film has been found to buffer the humidity inside the food package.

Showo Denko in Japan is manufacturing such type of duplex film under the trade name Pichit. CMC polymers based absorbents that form irreversible gels have been used to absorb the purge emanating from contained products like sliced and dried tomatoes, melons, watermelon, strawberries and chicken.

6) ODOUR REMOVERS

Many food products such as fresh poultry and cereal products develop the containment odours. Such odours may develop due to metabolic changes like formation of aldehyde or ketone compounds from lipid oxidation or anaerobic glycolysis and which are trapped within gas barrier packaging and when released are detected by the consumers. Activated carbon is a popular odour absorbent, which is effective and inexpensive. Vitamin E has also been aggressively marketed as a food grade odour remover in package materials.

Dupont has developed a series of molecular sieve compounds called odour and taste control, which reduces the off flavours arising from biochemical deterioration of some food products especially fatty foods such as potato chips.

7) ANTIOXIDANT RELEASING SYSTEMS

The major application of antioxidant releasing film is flavour protection of ready to eat dry cereals and noodles. Antioxidants must be "food grade" and must be transmitted from the film in appropriate rate under normal storage conditions to retard rancidity.

Several food grade antioxidants like butylated hydroxytoluene (BHT), butylated hydroxyanisole (BHA), vitamin E and dihydroguaric acid are being used.

BHT has been effective when added directly to dry cereals. When BHT was incorporated into HDP and enhance the shelf life of a contained oat cereal and also asserted that application of the antioxidants into the plastic film of the package were more effective than adding the same antioxidant directly into the contained food products. The antioxidants are incorporated on the interior surface of the film from a liquid solution which may be propylene glycol, glycerol or edible oil. The antioxidant in the solvent is trapped between the two films.

CHALLENGES AND REGULATORY ASPECTS:

This is beyond objection that seemingly introductory elements of active packaging that we are aware of today are going to be incorporated in next generation of food packaging. Isn't the packaging industry venturing too eagerly in unknown territories? The act of removing oxygen from internal package environment may be simple but it should be assured that contained residues do not overpower the scavenger and that the reacting compounds are neither toxic nor damaging to the contents. Furthermore, some active packaging systems have the potential to generate compounds capable of migrating into the contained food, eg - biocidal films are not inert and so must meet migration limitations (Brody, 2001a) So research needs to be done in these sundry aspects. Moreover, till now no specific regulations have been formulated to regulate the safety of active packaging and to promote the competitive positions in the industry.

CONCLUSION

Active Packaging is still developing as a collection of niche markets. So it is not surprising that a diverse range of Active Packaging systems which have been brought into light are destined to be magnum opus in the array of efficacious techniques for food preservation next to nonthermal processing. Intelligent combination of active packaging technologies with advanced food preservation techniques, material science, biotechnology and bioinformatics can extend the shelf life of food products while maintaining the nutritional, organoleptic and rheological quality and ensuring their safety.

Active packaging systems for the food industry would evolve as a new frontier in packaging and preservation of foods in the coming days. In Indian context it would be highly useful and potent system for extending the shelf life quality of a wide range of traditional dairy products such as paneer, halwa and sweet meats, etc. Although this packaging system is the latest one and in infancy stage but lot of research is required with great expectations used for food industry.

delay the ripening in fruits. Ethylene scavengers are used in fruits like banana, mango, grapes, etc. They are available in the form of sachets. Molecular sieves and silica gels are used to protect the food from harmful effects of moisture. Odour absorbers like activated Carbon, vitamin E are used in fried products like banana chips, potato chips to protect the natural flavour of foods. Antioxidants like vitamin E, Butylated Hydroxytoluene (BHT) and Butylated Hydroxyanisole (BHA) are used in noodles and ready to eat cereals to prevent oxidation and protect the flavour.

Active packaging is still in its infancy stage and in the future, it is bound to emerge as a concrete step in the evolution process of advanced packaging technologies to meet the requirement of innovative foods, provided that a few lacunae are taken care of and certain regulatory aspects are ascertained.

DISCUSSION:

1. Explain the functioning of Grape guard?

Grape guards are actually Sulphur pads. It is a brown sheet containing sodium metabisulphate (SMS). It produces sulphur gas, which absorbs carbon dioxide and other gases and prevents the fermentation and other deteriorative changes in the packages and thus it keeps the produce fresh.

2. What are the applications of biotechnology and bioinformatics in packaging?

Biotechnology and bioinformatics can be used to identify and develop antimicrobial compounds, natural antioxidants, etc. which can be safely incorporated into the packages.

3. What is shrink wrapper packaging?

It means wrapping the produce such as fruits or vegetables loosely inside a selectively permeable plastics film and sealed leaving volumes of air surrounding the produce. Whereas in individual seal packaging the film is shrink wrapped tightly around each piece of produce, which acts as another layer of cuticle.

4. How are antioxidants used?

The antioxidant substances are dissolved in solvents like propylene glycol and incorporated into plastic films and used for packaging the produce.

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KERALA AGRICULTURAL UNIVERSITY
COLLEGE OF HORTICULTURE
Department of Processing Technology

Hort. 651-seminar

Topic: Active Packaging

Name of student: Suja. G. Nair.

Venue: Seminar Hall
(College of Forestry)

Admission No. : 2004-12-12

Date & Time: 26-11-2005; 10.20 a.m

ABSTRACT

India is the second largest producer of fruits and vegetables in the world. The production of fruits is estimated to be 46 million MT and that of vegetables is 91 million MT. However, there exists a wide gap between the demand and supply of fresh fruits and vegetables due to the heavy post harvest losses. The increased production of fruits and vegetables will have significance only when they reach the consumer in good condition at a reasonable price. The existing postharvest losses of fruits and vegetables could be considerably reduced by adopting improved packaging, handling and efficient system of transport.

A package is a covering material, which should be capable of protecting the produce from the hazards of transport, preventing microbial and insect damage and minimizing physiological and biochemical changes. A variety of packaging materials such as boxes, cartons, glass bottles, jars, metal cans are available in the market. Modern concept of packaging involves aseptic packaging, C/M atmosphere packaging and active packaging.

The term 'Active packaging' was introduced by Labuza in 1987. It is defined as the group of technologies in which the package is actively involved with food product, or interacts with internal atmosphere to extend shelf life while maintaining quality and safety (Hloros *et al.*, 1997).

The potential active packaging technologies involve oxygen, carbondioxide and ethylene scavenging, antimicrobial packaging, moisture control, odour removers and antioxidant releasing systems (Prabha *et al.*, 2003). The oxygen scavengers, available in the form of sachets, films and labels, containing ferrous compounds, catechol, enzymes, etc. are incorporated in food packages to prevent off odour development, colour change, nutrient loss and microbial attack. Their main application come in vegetable oils, bakery products, dried fruits, cheese, etc. (Brody *et al.*, 2001). Carbondioxide scavengers, available in the form of sachets, containing calcium hydroxide or iron with calcium hydroxide are used to package fresh ground coffee and cheese. The major function of carbondioxide scavengers are to prevent the flavour loss of products. Antimicrobial packaging prevents the microbial deterioration of foods. Most widely used agents are silver salts on zeolite incorporated in plastic films and sheets. Organic acids, bacteriocins, spice extracts, enzymes, proteins, fungicides can also be used as antimicrobial agents in packages. Ethylene scavengers

are used in fruits like banana, mango and grapes to delay the ripening process. Potassium permanganate and activated carbon are generally used either in sachets, impregnated into labels or adhesives to trap the ethylene produced by ripening fruits or vegetables. Zeolite clays and silica gels are used in packages to protect the food from harmful effects of moisture. Odour absorbers like activated carbon, vitamin E are used in fried products like banana chips and potato chips to protect the natural flavour of foods. Antioxidants like vitamin E, Butylated Hydroxytoluene (BHT) and Butylated Hydroxyanisole (BHA) are used in noodles and ready to eat cereals to prevent oxidation and protect the flavour (Labuza and Breene, 1989).

Active packaging is still in its infancy stage and will emerge as a concrete step in the evolution process of advanced packaging technologies.

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